REPORTS

OF THE

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

VOLUME VIII.

Session of 1907-1908.

EDITOR:

GEORGE CARPENTER, M.D.

Hon. Secretaries:
LONDON:
J. PORTER PARKINSON, M.D. HUGH LETT, CH.B.
PROVINCIAL:
J. G. EMANUEL, M.D. (Birmingham).
ADDITIONAL FOR THE PROVINCIAL MEETING HELD IN BIRMINGHAM:
JAMES E. H. SAWYER, M.D. (Birmingham).

LONDON
J. & A. CHURCHILL
7, GREAT MARLBOROUGH STREET.

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OFFICE BEARERS AND MEMBERS OF THE COUNCIL OF THE SECTION FOR THE STUDY OF DISEASE IN CHILDREN OF THE ROYAL SOCIETY OF MEDICINE FOR THE SESSION OF 1908-1909.

ELECTED AT THE ANNUAL GENERAL MEETING.

Council (1908-1909).

Those marked thus (*) served on the Council of The Society for the Study of Disease in Children during the session 1907–1908, and those marked (**) served during the sessions 1907–1908 and 1906–1907; (†) Past Chairman of Council; (‡) Past Secretary; (‡‡) Past Provincial Secretary; (§) Past Honorary Editor of Reports (held office from 1900 to 1908); (§§) Past Honorary Treasurer (held office from 1900 to 1908); (‖) Held Office during the session 1907–1908; (¶) Wightman Lecturer for the session 1908–1909; (¶¶) Past Wightman Lecturer. For further particulars *vide* List of Members of the Section for the Study of Disease in Children of the Royal Society of Medicine in this volume.

*R. M. BEATTY, M.B. (Belfast).
¶§‡†GEORGE CARPENTER, M.D.
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‡†SYDNEY STEPHENSON, C.M.
*W. H. MAXWELL TELLING, M.D. (Leeds).
†A. H. TUBBY, M.S.
**G. L. TURNBULL, M.D.
*T. R. WHIPHAM, M.D.
‡‡**R. H. A. WHITELOCKE, C.M. (Oxford).

Honorary Secretaries.

LONDON:

‖HUGH LETT, Ch.B. E. I. SPRIGGS, M.D.

PROVINCIAL:

H. J. STILES, C.M. (Edinburgh).

LIST OF OFFICE-BEARERS OF THE SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN SINCE THE FORMATION OF THE SOCIETY IN THE YEAR 1900-1901.

Dates.	Chairmen of Council.	Honorary Secretaries.		Honorary Treasurer.	Editor of the 'Reports.'
		London.	Provincial.		
1900-1	A. E. Sansom, M.D.	Sydney Stephenson, C.M.	Charles H. Willey, C.M. (Sheffield).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1901-2	A. E. Sansom, M.D.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1902-3	A. H. Tubby, M.S.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1903-4	Sydney Stephenson, C.M.	George Carpenter, M.D. G. A. Sutherland, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1904-5	Sydney Stephenson, C.M.	G. A. Sutherland, M.D. Edmund Cautley, M.D.	R. H. A. Whitelocke, C.M. (Oxford).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1905-6	R. Clement Lucas, B.S.	Edmund Cautley, M.D. Francis Jaffrey.	R. H. A. Whitelocke, C.M. (Oxford)	R. Clement Lucas, B.S.	George Carpenter, M.D.
1906-7	R. Clement Lucas, B.S.	Francis Jaffrey. J. P. Parkinson, M.D.	Henry Skelding, B.C. (Bedford)	R. Clement Lucas, B.S.	George Carpenter, M.D.
1907-8	George Carpenter, M.D.	J. P. Parkinson, M.D. Hugh Lett, Ch.B.	J. G. Emanuel, M.D. (Birmingham). J. E. H. Sawyer, M.D. (Birmingham) (additional for the Provincial Meeting).	R. Clement Lucas, B.S.	George Carpenter, M.D.

RULES OF THE SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

NAME.

1. The name of the Society shall be "The Society for the Study of Disease in Children."

OBJECT OF THE SOCIETY.

2. The object of the Society shall be the study of disease and of allied subjects in children.

CONSTITUTION.

3. The Society shall consist of Ordinary, Temporary, and Honorary Members.

QUALIFICATION FOR MEMBERSHIP.

4. All duly registered medical men shall be eligible for membership.

OFFICERS.

5. The officers of the Society shall be elected from the ordinary members, and shall include an Honorary Treasurer, an Honorary Editor of 'Reports,' Honorary Secretaries (one of whom shall be resident in the London postal district, and one in some other part of Great Britain or Ireland), and twenty-four other members, of whom at least one third shall not be resident in the London postal district. These together shall constitute the Council and manage the affairs of the Society.

ORDINARY MEMBERS.

6. The names of candidates shall be proposed upon a printed form provided for the purpose, and signed by two or more members from personal knowledge. The names of candidates shall in the first instance be submitted to the Council, in which shall be vested the absolute power of declining to allow the

nomination to proceed. The proposal paper, after approval by the Council, shall be read at an Ordinary Meeting of the Society; and the ballot shall take place at the next meeting. No election shall be valid unless at least ten members vote, and no candidate shall be elected who fails to obtain four fifths of the votes recorded. No rejected candidate shall be eligible for proposal until twelve months have elapsed.

FORM OF ADMISSION.

7. Every Ordinary Member, after his election, and after payment of the annual subscription, and of the entrance fee (if any), shall sign the roll of members, declaring thereby his adhesion to the Rules of the Society.

TEMPORARY MEMBERS.

8. Any resident medical officer of a hospital or infirmary shall be eligible for election by the Council as a Temporary Member of the Society for a period of twelve months, without payment of any subscription or entrance fee. He shall be entitled to attend the ordinary Meetings, but he shall not have a vote, shall not be eligible for office, shall not be entitled to receive a copy of the 'Reports,' and shall not introduce visitors. If, at the termination of the period for which he has been elected a Temporary Member, he desires to become an Ordinary Member of the Society, he must go through the usual form of election, but shall be exempt from paying any entrance fee.

HONORARY MEMBERS.

9. The Council shall have power to propose for election as Honorary Members the names of persons eminent in medicine, surgery, or the collateral sciences. The Honorary Members, who shall not exceed twelve in number, shall be balloted for, after proposal by the Council, at a General Meeting of the Society, and be subject to the same rules as govern the election of ordinary members. They shall enjoy all the privileges of ordinary members, voting excepted. They shall not be called upon to pay any fee or subscription.

RESIGNATION AND EXPULSION OF MEMBERS.

10. Any member may retire from the Society on giving written notice of the intention to do so to one of the Secretaries and on paying any contributions that may be due.

The name of any member may be expunged from the Society's roll only at a General Meeting convened by the Council specially for consideration of the case. At least ten votes must be recorded, and four fifths of the votes, if recorded in favour of the expulsion, shall be decisive. Any person expelled in accordance with this rule shall forfeit all claim upon the Society.

Subscriptions.

11. The annual subscription for Ordinary Members shall be One Guinea, payable in advance at the beginning of the session in October. Ordinary Members elected after July the 31st, 1901, shall pay an entrance fee of One Guinea; but in the case of any member elected subsequently to Easter in any year the member shall not be called upon to pay a subscription during the ensuing session. Original Members pay no entrance fee. A member shall at any time be exempted from further annual subscriptions by paying a composition fee of Ten Guineas.

A member whose subscription is six months in arrear shall be reminded of the fact by one of the Honorary Secretaries; and if the amount due remain unpaid during the current year, his name may be removed by the Council from the list of members.

Election of Officers.

12. The Officers of the Society shall be elected by ballot at the Annual Meeting. No member, except the Honorary Treasurer and the Honorary Editor of the 'Reports,' may hold the same office for a longer period than three consecutive years. A list of names recommended by the Council for election shall be sent to each ordinary member at least fourteen days before the Annual Meeting of the Society. A name or names may be substituted on the voting paper for that of any member or members proposed by the Council for election. Two Scrutineers shall be appointed by the Chairman at the commencement of the Annual Meeting, and their duty shall be to receive and examine the balloting lists, and report the results to the meeting. In the case of equal voting the Chairman shall decide by lot.

Honorary Treasurer.

13. The Honorary Treasurer shall have charge of the funds of the Society, and keep an account in an orderly and business-like way of all sums that pass through his hands. He shall receive all moneys due to, and pay all moneys due from, the Society, only after the accounts have been passed by the Council and signed by two of the members present. He shall keep a printed receipt-book for subscriptions and fees, and every receipt shall be signed by him and countersigned by one

of the Honorary Secretaries. He shall prepare an annual statement of accounts, which shall be audited by two ordinary members of the Society not in office, and presented, with the Council's Report, to the next Annual Meeting. He shall keep a separate banking account in the name of the Society, and shall invest the funds from time to time as directed by the Council. The Honorary Treasurer shall be *ex officio* a member of Council.

HONORARY EDITOR OF 'REPORTS.'

13A. The Honorary Editor of the 'Reports' shall edit the Society's 'Reports' and he shall keep all papers and communications and illustrations intended for publication in the 'Reports.' He shall be *ex-officio* a member of the Council.

HONORARY SECRETARIES.

14. The Honorary Secretaries (or one of them) shall conduct all correspondence, notify to new members their election, and convene, attend, and minute all meetings of the Society, of the Council, and of any committees appointed by the Council. They shall arrange the order of proceedings at all meetings. They shall submit to the Council all official communications and all nominations for membership in the Society. They shall keep a register of all cases and papers communicated to the Society. They shall keep a roll of the officers and members of the Society. In the discharge of these various duties the Secretaries shall have power to employ clerical assistance, to use printed or typed forms, and to prepay postages and telegrams. They shall be *ex officio* members of Council.

MEETINGS.

15. These shall be *Ordinary*, *Special*, and *General*.

Ordinary Meetings shall be held at hospitals in London or elsewhere, or in places appointed by the Council. There shall not be more than eight Ordinary Meetings in the year, and of these one at least shall be held elsewhere than in London. The meetings shall take place upon the third Friday in the months of October, November, January, February, March, April, May, and June, at 5.30 p.m., unless otherwise arranged by the Council. They shall not be of longer duration than two hours, except by special resolution of the Meeting. Clinical and Pathological Demonstrations, and discussions thereon, shall take precedence over papers. Nothing relating to the laws or the management of the Society shall be discussed at an Ordinary Meeting.

Special Meetings may be convened by the Council for the purpose of discussing a subject or subjects deemed to be of particular interest in relation to disease in children, or to allied subjects.

A *General Meeting* shall be called by the Honorary Secretaries on the requisition of any five members of Council or of any twenty ordinary members of the Society. At least fourteen days' notice shall be given to every ordinary member of the Society; and the character of the business to be transacted at such meeting shall be stated on the summons paper. No business other than that set forth upon the summons paper shall be considered at the meeting.

The *Annual General Meeting* shall be held at a time and in a place determined by the Council, fourteen days' notice having been given to every ordinary member of the Society. Its business shall include election of officers, presentation of the annual Report of the Council, and the discussion of any proposed alteration or alterations in the rules (notice of which shall be given in the summons convening the meeting). Ten ordinary members shall form a quorum. No change in the rules shall be valid unless three fourths of the votes are recorded in favour of the alteration. A notice, written or printed, may be served upon any member, either personally or by sending it through the post in a prepaid letter, addressed to such member at his registered address.

CHAIRMAN.

16. A Chairman, who must be an ordinary member of the Society, shall be chosen by show of hands or by ballot at the commencement of each meeting, whether ordinary, special, or general. He shall sign the minutes of the preceding meeting or meetings, regulate the proceedings, check irregularities, interpret the application of the laws, and enforce the recognised rules of debate. The Chairman shall have an ordinary and a casting vote.

PUBLICATIONS.

17. Reports shall be published in such manner and at such times as the Council may direct. A copy shall be forwarded free to every Honorary Member of the Society, and to every ordinary member whose subscription is not in arrear.

VISITORS.

18. An ordinary member may introduce two medical men as visitors at ordinary meetings of the Society. Visitors shall sign their names in the book provided for the purpose. On the invitation of the Chairman, visitors shall be permitted to take

part in the discussions of the Society. The same visitor shall not be introduced more than twice during one session.

COUNCIL.

19. The Council shall have the management of the affairs of the Society, and shall meet as often as may be necessary for the transaction of business. Seven days' notice of each meeting shall be sent by the Honorary Secretaries to every member of Council. Five members shall form a quorum. The Council shall determine questions, if necessary, by ballot. The Council, at its first meeting, shall elect from amongst its members a Chairman, to preside over its deliberations during the session. He shall not hold office for longer than two consecutive years. In his absence the chair shall be taken by some other member of Council, duly proposed and seconded The Chairman of Council shall have a casting, in addition to an ordinary vote. The Council shall from time to time make regulations, issue instructions, appoint committees, and take any other lawful steps to further the good government of the Society. It shall prepare and present to the Annual Meeting a Report upon the state of the Society. The Council shall be empowered to fill all vacancies that may occur from death or otherwise between one Annual Meeting and another. It shall decide all questions relating to the publication of the 'Reports.' It shall annually depute three of its members, with the Honorary Secretaries, to form a *Committee of Reference*, the duty of which shall be to determine the acceptance of communications referred to it by the Honorary Secretaries. The Council shall be elected by ballot at the Annual Meeting. One third of its members, exclusive of the Honorary Treasurer, the Honorary Editor of the 'Reports,' and the Honorary Secretaries, shall be replaced annually by an equal number of ordinary members of the Society. An Extraordinary Meeting of Council may be called (1) by the Chairman of Council, or (2) by the Honorary Secretaries, and (3) shall be called by the Honorary Secretaries on the requisition of any five members of Council. Seven days' notice of such extraordinary meeting must be given in writing by the Honorary Secretaries to every member of Council upon a summons paper specifying the object for which the meeting is called.

SUGGESTIONS FOR PROVINCIAL MEETINGS.

1 These meetings are arranged by the Provincial Secretary, who will be assisted and advised by the London Secretaries, and the latter with the Council will control all details.

2 Meetings within one hundred miles of London are held on Saturdays

3. The Secretaries should send out a preliminary notice of the Provincial Meeting with the announcement of the Wightman Lecture

4 In the notice calling the Meeting indication should be given where lunch can be obtained, but this meal should be quite informal. On this notice the most convenient trains to and from the place of meeting should be stated.

5 Prior to the formal opening of the proceedings there should be a demonstration of cases in the wards of the hospital or other suitable place, and an abstract printed or type-written of the more important cases to be demonstrated should be distributed among the members. This usually commences at 2.30 or 3 p.m.

6. A Museum of Pathological Specimens should be provided, all of which should have attached adequate descriptions, either type-written or printed.

7. Two hours should be allowed for the discussion of papers.

8. The Chairman of the Provincial Meeting should be the Chairman of the Council.

9. There should be an adjournment for tea at a time to be arranged, for half an hour, either between the clinical demonstration and the reading of papers, or during an interval in the time devoted to the latter.

10. In the evening there should always be a public dinner to which guests, not necessarily medical men, may be invited. The Chairman should be a local medical man, or a member of the Society nominated by the Local Organising Secretary, to be approved by the Council.

11. The Secretary should arrange that the charge for each

dinner ticket should be rather more than the contract price of the dinner, so that all expenses may be covered without drawing on the funds of the Society.

12. The Local Secretary should do his utmost to awaken the interest of all members of the profession residing in the town or neighbourhood in the Provincial Meeting, and should persuade as many as possible to become members either before or during the meeting.

13. He may strengthen his position, if he so desire, by forming a local committee to assist him in carrying out his programme.

MEETINGS BEYOND A RADIUS OF A HUNDRED MILES FROM LONDON.

If the Provincial Meeting be arranged at a distance of more than a hundred miles from London, it may be called on a Friday instead of Saturday.

The programme for the first day should then be the same as that previously stated.

On Saturday morning arrangements may be made for the members to visit the hospitals or places of interest in the city or town. In the afternoon an excursion may be arranged to some neighbouring place of interest, the expenses of which must be defrayed by those joining it.

REPORT OF THE COUNCIL, 1907–8.

Read at the Annual General Meeting, held at No. 11, Chandos Street, Cavendish Square, W., on Friday, July the 17th, 1908.

THE Council have the pleasure to report that the prosperity of the Society is well maintained, though a certain number of members have resigned owing to the Society having not yet joined the Amalgamation of Medical Societies, yet so many new members have been elected that the roll has slightly increased.

The meetings have been well attended and many interesting cases, specimens, and papers have been brought before the Society and discussed.

A special meeting was held in December for the discussion of "Inherited Syphilis," and the Wightman lecture was delivered by Sir W. Watson Cheyne, Bart., on "The Defensive Arrangements of the Body, as illustrated by the Incidence of Disease in Children and Adults."

Dr. George Carpenter has been appointed Wightman Lecturer for the session 1908–1909.

The Provincial Meeting was held in Birmingham on June the 20th, and was attended by about 150 local medical men as well as by the members, who were received at the General Hospital by Sir Thomas Chavasse, Senior Surgeon to the Hospital, and the Chairman of Council, Dr. George Carpenter. A large number of interesting cases and specimens (both naked-eye and microscopical) were exhibited at the General Hospital, and following this a very well attended meeting was held in the Out-Patient Hall of the Children's Hospital, the chair being taken by the Chairman of Council. In the evening the members and visitors dined at the Queen's Hotel, Sir James Sawyer occupying the chair.

The meeting was a very successful one, and the Council desire to express their indebtedness to Dr. James E. H. Sawyer, who undertook the local arrangements owing to the absence of Dr. Emanuel, and to those members of the profession in Birmingham who took part in providing the programme.

The next Provincial Meeting will take place in Edinburgh.

At a meeting of the Council on July the 8th it was recommended that the Society join the Royal Society of Medicine as the Section for the Study of Disease in Children.

The Society regrets the death of three members: Dr. Henry Ashby, Professor of Diseases of Children at the Manchester University, Dr. Bertram Abrahams, Assistant Physician at the Westminster Hospital, and Mr. H. J. Prangley, of Anerley. Dr. Ashby was an original member of the Society and took the greatest interest in its welfare; he read many valuable papers at its meetings. A letter of condolence has been sent to his widow by Dr. George Carpenter, the Chairman of the Council.

TREASURER'S REPORT.

The Hon. Treasurer reports with pleasure the continued financial stability of the Society and the gradual increase in its capital.

During the past year £100 Consols have been purchased, bringing up the total invested capital to £800 Consols, a position which no similar society appears ever to have reached in the same number of years. After discharging all debts there remains a balance at the bank of £100. 15*s.* 1*d.* Owing to the late appearance of the

'Reports,' due to the fact that contributors did not promptly supply the Editor with their MS, the notice reminding members that their subscriptions were due was not sent out till after Christmas, and as a consequence a good many subscriptions are still uncollected.

From the Wightman Lecture Fund £20. was handed to Sir W. Watson Cheyne, Bart., for his lecture, and there remains in hand a balance of £20. for next year's lecture.

R. CLEMENT LUCAS.

THE ANNUAL GENERAL MEETING.

The Annual General Meeting was held at 11, Chandos Street, W., on Friday, July the 17th, at 5 p.m., the Chairman of Council (Dr. George Carpenter) in the chair. There were present: Mr. R. Clement Lucas (Treasurer), Dr. Percy G. Lewis (Folkestone), Dr. Frederick Taylor (Trustee), Dr. J. Porter Parkinson (Secretary), Dr. F. W. Higgs, Dr. Leonard Guthrie, Mr. J. Scott Battams, Dr. E. I. Spriggs, Dr. Edward J. Nix, Dr. L. A. Clutterbuck, Mr. Russell Howard, Dr. C. O. Hawthorne (Member of Council), Mr. Hugh Lett (Secretary), Dr Sidney Gilford, Dr. T. R. Whipham (Member of Council), Dr. W. Ewart, Dr. G. L. Turnbull, Dr. Joseph B. Wallace, and Dr. George Pernet.

The following business was transacted: Several new members were balloted for and declared duly elected members of the Society. The office bearers nominated by the Council for the session 1908–1909 were then balloted for, Mr. Yearsley and Dr. Hawthorne being appointed scrutineers by the Chairman, and the nominations were unanimously confirmed.

Having received the report of the Council and having listened to the Treasurer's statement, the question of amalgamation with the Royal Society of Medicine was then taken.

The Chairman (Dr. George Carpenter) said: For the information of those members who were not able to be present at the dinner held at the Provincial Meeting in Birmingham, it affords me much pleasure to announce that the members then present, on hearing of Dr. Henry Ashby's illness, sent him a telegram of sympathy, wishing him a speedy convalescence, and that I received in reply the following acknowledgment:

"13, St. John Street,
Dictated "Manchester,
"*June* 21*st*, 1908.

"My dear Carpenter,

"I was very much gratified to receive the telegram this morning you were good enough to send off last night.

"It is a great pleasure to think that one is not forgotten by one's friends when one is laid by. Will you thank the members of the Society for their thoughtfulness and good wishes.

"Yours very sincerely,
"H. A., (*for*) Henry Ashby."

The special notice (herewith appended) has been in the hands of members for some time, and I do not suppose they wish to have the terms upon which amalgamation has been discussed between the Council of the Society and the Royal Society of Medicine read over to them, but I shall be glad to hear the views of the members present on this, to them, vital question.

SPECIAL NOTICE.

At a meeting of the Council, held on July the 8th, 1908,* it was unanimously decided to recommend that on the strength of the proposals made to this Society on behalf of the Royal Society of Medicine by their Secretary, that The Society for the Study of Disease in Children should join that Society as the "Section for the Study of Disease in Children," subject to the undermentioned terms, agreed upon between them, being confirmed by the Royal Society of Medicine.

This recommendation will be laid before the members of the Society at the Annual General Meeting, and the matter will be decided by the votes of the members present, a three-fourths majority being necessary before any change can be effected.

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* At the meeting of the Council a report of the Sub-Committee, consisting of Dr. George Carpenter (Chairman of Council), Mr. R. Clement Lucas (Hon. Treasurer), Dr. Porter Parkinson (Hon. Sec.), Mr. Hugh Lett (Hon. Sec.), Dr. G. A. Sutherland and Dr. Edmund Cautley, appointed by Council to meet the Secretary of the Royal Society of Medicine to discuss terms of amalgamation, was presented.

The terms of amalgamation which have been agreed upon are as follows:

1. That all members of The Society for the Study of Disease in Children be admitted into the Royal Society of Medicine and shall form a section to be called the "Section for the Study of Disease in Children," without entrance fee, and with the option of becoming Fellows of the Royal Society of Medicine if they so desire, without entrance fee.
2. That all life members shall become life members of the Section.
3. That the Chairman of the Council of the Section or his representative shall be the representative of the Society on the General Council of the Royal Society of Medicine.
4. That the Provincial Meetings be provided for as heretofore.
5. That the members of the Section shall have the following options with regard to the 'Transactions':
    - *α*. That a fasciculus of the 'Transactions' of the Section be delivered monthly, which may be returned to be bound at the end of the year at a cost to the member of *2s. 6d.*
    - *β*. That a bound volume of the 'Transactions' of the Section be delivered annually without extra cost for binding.
6. That the Section has absolute power as to the publication of the material brought before it.
7. That previous to amalgamation all expenses of the Society incurred during the present Session shall be discharged, including that of the volume of 'Reports' now in hand.

Mr. R. Clement Lucas said: As I occupied the chair when this subject was discussed last year, and held a brief on the part of the Council to oppose the amalgamation scheme at that time, I should be glad of an opportunity of explaining why I am now prepared to advise the Society to join the Royal Society of Medicine.

My criticisms, it may be remembered, were chiefly directed against the financial aspect of the scheme, as then represented, which I maintained was unsound; a prospective balance of £1200, after maintaining eighteen societies as sections, I held was too narrow, and I especially drew attention to the fact that the cost of publishing for these societies had been altogether under-estimated. not only was this the case in the matter of print, but an even greater error had been made as regards illustrations. The amalgamation of societies has now been twelve months in existence, and it has been found, as I predicted, that their greatest loss has been under the head of publication. It may be asked why did I argue against amalgamation in this Society and vote for amalgamation in others? The answer is easy. The three other societies to which I belonged were rapidly declining. One was hopelessly bankrupt, and the other two were unable to meet their annual liabilities out of their annual income. Interest in their meetings was also rapidly waning. For them amalgamation was the only remedy against dissolution. But this Society was on an entirely different footing, and could well afford to wait. Its financial position was unassailable, its expenditure was well within its income, and it was rapidly accumulating useful capital, so that in less than eight years it had an invested capital of £800 Consols. It would have been gambling to risk such a position on insecurity. The resolution, however, which we passed at the general meeting last year did not close the door to reconsideration. It was: "That The Society for the Study of Disease in Children declines, under present circumstances, to join in the scheme of amalgamation." Now, last year it was impossible to join in the amalgamation without a radical alteration in our constitution, for no provision was made for any but a president to act on the General Council of the Royal Society of Medicine, nor was there any provision for country meetings, or for other than monthly unbound 'Proceedings.' In all these matters the Royal Society of Medicine is willing to meet us. The representative need not be a president, country meetings will be provided for as before, and there will be an option to have either monthly 'Proceedings,' which can be bound for 2*s.* 6*d.*, or a yearly bound volume as at present. Our members enter without entrance fee, or may become Fellows on similar terms. Life members will not have to pay so much for their fellowship, as the compounding fee is taken into consideration in the subscription.

The Royal Society of Medicine has done us the honour of imitating several of our customs. Several of the sections now meet in the afternoon instead of evening, and this has been found to largely increase the numbers of those attending. Some sections now hold country meetings, and several have followed us in holding one meeting a month, instead of two. I understand that we shall be able to meet on the same day and at the same hour as hitherto, and that we shall have complete control over our publications, so that there seems nothing now that we can raise objection to. What we have done cannot be undone. We have compelled the recognition of the special study of disease in children. The Royal Society of Medicine is now firmly established. It was calculated in the circular which was sent out that they would only have 1000 Fellows, whereas now they have something like 2700 Fellows. Of course, after calculating on a basis of 1000 Fellows, to have 2700 Fellows, each paying three guineas, places the whole financial position of the Royal Society in a different position, and a very different one from what it was estimated to be at the beginning. Taking the whole of these things into consideration, I think this Society would be wise in joining the amalgamation, because we do not want to divide those who are interested in children's diseases; we want to heal any dispute or discontent. The object of this Society, I maintain, is one, and a simple one, and that is to do our very best in the interests of the study of disease in children. (Applause.)

The CHAIRMAN (Dr. GEORGE CARPENTER): I will call upon Dr. Porter Parkinson to read a letter which the Council received on July 9th from the Secretary of the Royal Society of Medicine in regard to the terms of amalgamation.

Dr. PORTER PARKINSON read the letter as follows:

"20, Hanover Square, W.
"*July* 10*th*, 1908.

"DEAR SIR,

"*Society for the Study of Disease in Children.*

"I am very glad to be able to inform you that at the Council meeting just over, the proposals of your Society were unanimously accepted, and the necessary resolutions passed for creating the new Section.

"Everything, therefore, is now in order, and if your Society passes the necessary resolutions on the 17th, the officers of the two Societies can immediately proceed to carry out the details, with a view, if possible, of having everything in readiness for beginning next Session under the new conditions.

"Faithfully yours,
"J. Y. W. MACALISTER, *Secretary.*"

Dr. J. PORTER PARKINSON,
57, *Wimpole Street, W.*

Mr. R. C. LUCAS: I do not know, sir, whether you intend to move the resolutions from the chair.

The CHAIRMAN (Dr. GEORGE CARPENTER): Yes, that is my intention.

Mr. LUCAS: I suggest that the Council are of opinion it would be well to pass a resolution that the Society is in favour of amalgamation, provided these conditions which have been circulated are sanctioned by the Royal Society of Medicine, and then that it should be given to a small committee to wind up the affairs of the Society; and that a special general meeting should be called early in September to dissolve the Society. If these resolutions were put from the chair it would be the simplest way of dealing with the matter.

Dr. CHARLES W. CHAPMAN: The first part of September is rather early, Mr. Chairman, and many people will be away then. If you could put off the general meeting until the middle of September it would be much better.

The CHAIRMAN (Dr. GEORGE CARPENTER): It would be somewhat inconvenient to have it early in September, because I do not know whether it will be possible to produce the 'Reports' by that time. I will do my best, however, in this respect, but if it could be postponed to the end of September I think it would aid the work of the officials. There is an exceedingly large amount of work to be done before the 'Reports' can be issued to Members, and if the volume is ready even by the end of September it will necessitate work at high pressure and in the vacation. I propose the first resolution, and that is:

"That, subject to the conditions circulated, the Society agrees to the scheme of amalgamation."

Dr. C. O. HAWTHORNE: Sir, before you put that motion I should like to say a few words upon this subject, as one who has taken part in the discussion of this question upon a former occasion. On that occasion I appeared as a member of a small and feeble, if not despised, minority. By a curious turn of affairs, it appears that to-day we are in a position of happy unanimity. Well now, sir, in those circumstances the minority, I am quite sure, is not in the least anxious to make any claim for superior prescience, nor is it anxious to discuss the question whether gentlemen who voted in one direction upon a former occasion and have announced their intention to vote in the exactly opposite direction on the present occasion—I say the former minority is not anxious to discuss how far that change in position has been brought about by a change of view on their part, or by an alteration in the policy of the Royal Society of Medicine. I am sure I shall speak for those who voted with me on a former occasion when I say that we are more than content to find that either a change of mind or a change of circumstances—we will not inquire which—has brought us into a position to-day in which the Society is likely to assent unanimously to this resolution of the Council. There is, however, one voice that I think we might have looked for at this supreme moment, a voice which I expected would be one of warning, namely, that of Dr. Pernet. Some of us well remember that on a former occasion he appeared as the master and the champion of the art of gloomy and pessimistic prophesy, and he warned us that anything like a union of societies would infallibly loosen the knees of our secretaries and other officials, and that great institutions, instead of promoting vigour and unanimity, would lead to lethargy and decay. I had hoped and expected that at this moment we should at least have heard from that quarter that these dangers are not affected by any alteration in the financial proposals. There fell upon my devoted head, on the occasion to which I refer, the eloquent vials of Dr. Pernet's wrath, and that he is silent to-day I take it we all regard as a happy augury. I, for one, cordially support the resolution from the chair.

Dr. George Pernet: I am very glad Dr. Hawthorne has mentioned my name. I was waiting for Dr. Hawthorne to address us, because my experience has been at former meetings of The Society for the Study of Disease in Children that Dr. Hawthorne has always got up and said something which was against everybody else. I expected that he would now take a turn in the other direction, and denounce amalgamation from head to foot. But I would point out to Dr. Hawthorne that now, instead of being whipped in like children, we are going in with the drums beating and colours flying. We have obtained our conditions, and if we had given in at the time Dr. Hawthorne was so anxious for us to join the scheme, we should have been a Section, we should have had a President in the usual way, our 'Transactions' would have been tabooed, and perhaps even our Provincial Meeting would not have been permitted by the gentlemen at Hanover Square. I am a Fellow of the Royal Society of Medicine myself, and therefore cannot be suspected of any personal bias, but now we have been offered terms, and we ensure that the traditions of The Society for the Study of Disease in Children will be carried on in a way which would not have been done if we had given in at the beginning. We know where we are, and although I am willing to work as loyally for the Section as I have done for the Society, and always intended to do so, I must, however, point out that, from my knowledge of the working of the Sections at the Royal Society of Medicine, there may be difficulties. For instance, the Section of Dermatology is becoming unpleasantly overcrowded. We shall not be able to keep anybody out of the Section, and we may be a bit overcrowded in consequence, but we shall have to put up with it. I shall certainly support this resolution, because, as I say, we have been offered acceptable terms and we should not have been offered them if we had gone in straight off.

Dr. William Ewart: Perhaps some of the members at this meeting would like to know what arrangements have been made financially, what equivalent this Society gets for the respectable amount of, I think it is, £800, which I suppose will be handed over to the coffers of the Royal Society of Medicine? What equivalent do we get for that compared with the equivalent obtained by a bankrupt society, or a society on the verge of bankruptcy, some of the others which were mentioned? They get advantages, and we get advantages; they pay nothing, we pay £800. Perhaps some of the members will be interested, as I am, in knowing what is to represent that value to us, whether we have secured a *quid pro quo* to the amount of that? With regard to the main question of amalgamation, I have still a kind of idea—though of course the Council have discussed these things and are *au fait* with them—that as an independent society with some financial support we hold a prouder and better position, certainly a more unique position, than we shall hold afterwards when we are merged. There is no doubt that several of us a year ago, when this question was discussed, thought we had an asset which we could not sacrifice lightly, and that was the asset of our independence and energy as a separate society, with perfect freedom for any developments which we chose to undertake. All this falls from us now because unless we get an equivalent for the sum which we contribute, some equivalent in privilege, we give up the privilege of freedom and give up the privilege of possible development along lines of our own. What we may do in the future, additional work besides urging the study of disease in children, what we may possibly do will have to be done for us by the Royal Society; we shall have no real action in the matter, we shall be under their orders. Therefore, sir, there at once falls away the dream of ambition, the ideals on a large scale which have floated before the eyes of some of us. At the same time, there are ideals which are worth pursuing, and there was that of the study of childhood from the point of view of disease. There was a big opening there, and it seemed an object which this Society seemed especially fitted to undertake. Therefore I cannot help personally having a regret that this individuality is going to be wiped out. I also am a member of the Royal Society, and I am proud of its developments and of its growth, and of its members and its officials particularly. But nevertheless, here is a thing which was, and is going to cease to be. It had possibilities in the future which were great, but it is to be absolutely removed from our horizon. I do not think I can consistently vote for the expunging of this Society, although I am sure it will do admirable work and will have many advantages which it would not have otherwise by joining the Royal Society of Medicine. But I do not see how it is going to do its inimitable work as it otherwise would do.

The Chairman (Dr. George Carpenter): In answer to your inquiry, Dr. Ewart, the Council did not look on this matter as a simple question of £800. What was thought was this, that it was in the interests of the children that amalgamation should take place.

Dr. Frederick Taylor: Dr. Ewart might be reminded, I think, that it is stated in the very first law, or bye-law or regulation—I have not it before me—that the object of the Society was to promote the study of disease in children, not to accumulate money, even to the extent of £1000. Now there is this money accumulated we ought to do something with the money, or make the subscription less. The object of this Society is to promote the study of disease in children. We did so by joining the Society, and we should be equally ready to do so by either dissolving the Society or by joining ourselves to the Royal Society of Medicine. As an associate with Dr. Hawthorne last year on the question, I may say that I have had that point of view throughout the year that is past.

Dr. Leonard Guthrie: I should like to mention that a year ago, at the discussion on this subject, several of us drew attention to the fact that a Section for the study of disease in children was bound to come, and as a matter of fact that Section has not been formed up to the present mainly, I think, with the view of inducing us to become the Section ourselves. If it had been formed, and we stood out, I venture to predict that the death of this Society would speedily occur, and I note with extreme satisfaction that we are to amalgamate.

Dr. Porter Parkinson: It seemed to me all along that the crux of the whole matter was that £800. Some weeks ago I suggested to the Treasurer a very reasonable way of getting rid of that £800 would be on the lines of the Royal College of Physicians: that we might have a dinner, and that a certain amount might be distributed in that way. But I am afraid that that suggestion was rather sat upon at the time.

The Chairman (Dr. George Carpenter): Are there any dissentient voices, other than those which have just been expressed? No? Then the first resolution is: "That, subject to the conditions circulated, the Society agrees to the scheme of amalgamation."

Carried unanimously.

The CHAIRMAN (Dr. GEORGE CARPENTER): The second resolution is: "That the Standing Committee shall be authorised to bring out the 'Reports' and to close the financial business of the Society previous to amalgamation." I may say that the Standing Committee is the present Chairman of Council, the Treasurer and the Secretaries.

Carried unanimously.

The CHAIRMAN (Dr. GEORGE CARPENTER): The third resolution is: "That a special general meeting be called to dissolve this Society;" we will say in September.

Carried.

Dr. PERNET: That means subject to these conditions.

The CHAIRMAN (Dr. GEORGE CARPENTER): Do you want that put in?

Dr. PERNET: I think so; it makes it clearer: "Subject to the conditions being fulfilled by the Royal Society of Medicine."

Agreed.

The CHAIRMAN (Dr. GEORGE CARPENTER): That concludes the business, gentlemen.

THE HISTORY OF THE SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

It was a striking fact that in this country no effort had been made to systematise the study of affections of children, or to evoke the free interchange of experience by open discussion until 1900. London and many of the provincial cities are well equipped with children's hospitals, yet either the energy was wanting or the need of such a Society was not understood. And this is all the more striking when we know that in France, Germany, and the United States not only were there ample opportunities for the interchange of views, but special journals had been founded to deal with the problems of illness in childhood, and had proved successful both from the scientific and the financial standpoint. As far back, however, as 1896, an attempt was made in this country to evoke a greater interest in children's diseases by the publication of an Anglo-American Journal, 'Pediatrics,' dealing with this subject. Men, some of whose names frequently recur in this account, supported it by their literary contributions, viz. Dr. Henry Ashby, Dr. Leonard Guthrie, Mr. Sydney Stephenson, Dr. G. A. Sutherland, Mr. Macleod Yearsley, Mr. Edmund Owen, Dr. John Thomson, Dr. Wayland Chaffey, Mr. Charles Morton, and others, but the psychological moment had not arrived, and the venture succumbed, as far as the English edition was concerned, in a comparatively short time. Dr. George Carpenter, the English editor, however, was not daunted, and in 1904 founded the 'British Journal of Children's Diseases.'

So little was this anomalous situation appreciated that at the annual meetings of the British Medical Association sometimes a children's section was given a place on the programme, but often not. Indeed, some few years before the Society's foundation, it was the boast of a well-known man attached to an equally well-known children's hospital in London that there was no such thing as a children's specialist in London. And, further, it must be understood that those in medical authority, although they acknowledged children's hospitals, and countenanced consultants using these institutions as stepping-stones to appointments at general hospitals, yet the special study of children's complaints was not viewed with favour, and the cause of the children greatly suffered in consequence.

The idea of forming a society for the study of disease in children originated with Mr. Alfred H. Tubby and Mr. Sydney Stephenson, who, towards the end of May, 1900, sent a circular to members of the honorary medical and surgical staffs of the children's hospitals in London and the Provinces, inviting them to attend a preliminary meeting at 20, Hanover Square, W., at 5.30 p.m., on Monday, July the 11th, and failing attendance to offer suggestions and state whether they were in favour of the promotion of such a society, and, if so, were they willing to become members. The preliminary meeting was held and Dr. A. Ernest Sansom took the chair.

About sixty medical men attended, the majority being whole-hearted in their wishes for the success of the project; some were sitting on the fence, and a small minority were openly antagonistic. It is interesting now to recall that the signatories to the original letter had been entreated by means of lengthy epistles not to go on with their "ill-conceived project," but to abandon it forthwith as it was doomed to failure. Another interesting incident is that shortly after the project had been publicly mooted, one of the originators was favoured by a visit from a physician eminent in this special branch of work, who said he had received the letter, but before he answered it he wished to know "if the Society were respectable." Happily the writers of the letter were able to assure him that their wishes and intentions were entirely altruistic, and all that they desired was to help in the advancement of the study of disease in children. And when the Society had been in existence for a number of years, a consulting surgeon attached to one of the London children's hospitals was invited by a colleague at the same medical school to join the Society, it is amusing to relate that in reply this surgical Rip Van Winkle had never heard of the Society.

However, it was unanimously agreed to form a society for the study of disease in children and a Provisional Committee was appointed. The Provisional Committee consisted of Mr. Henry G. Armstrong (Wellington College), Dr. Henry Ashby (Manchester), Dr. Wayland C. Chaffey (Brighton Hospital for Children), Dr. Arthur Downes (Medical Inspector, Local Government Board), Mr. Walter Edmunds (Evelina Hospital for Children), Dr. Theodore Fisher (Bristol Hospital for Children), Mr. Jonathan Hutchinson (London Hospital), Dr. David B. Lees (Great Ormond Street Hospital for Children), Mr. R. Clement Lucas (Evelina Hospital for Children), Mr. Howard Marsh (Great Ormond Street Hospital for Children), Dr. Lewis Marshall (Nottingham Hospital for Children), Dr. John McCaw (Belfast Hospital for Children), Mr. D'Arcy Power (Victoria Hospital for Children), Dr. Humphry D. Rolleston (Victoria Hospital for Children), Dr. A. E. Sansom (North-Eastern Hospital for Children), Dr. George E. Shuttleworth (Royal Albert Asylum for Imbeciles, Lancaster), Mr. Harold J. Stiles (Edinburgh Hospital for Children), Dr. George A. Sutherland (Paddington Green Hospital for Children), Mr. Alfred H. Tubby (Evelina Hospital for Children), Dr. Dawson Williams (East London Hospital for Children, Editor 'British Medical Journal'), and Mr. Sydney Stephenson (Evelina and North-Eastern Hospitals for Children) (Hon. Sec. *pro tem.*).

A sub-committee was appointed for the purpose of drafting the constitution of the Society, which consisted of Dr. David B. Lees, Dr. A. Ernest Sansom, Dr. George A. Sutherland, Mr. Sydney Stephenson, and Mr. Alfred H. Tubby, and the proposed constitution having been submitted to the Provisional Committee, the suggested rules* were laid before an Inaugural Meeting of the Society, held on July the 24th, 1900, at 20, Hanover Square, and adopted.

In passing it may be mentioned that Mr. Tubby and Mr. Stephenson had had an informal discussion as to the lines on which the future society should be asked to run. It was felt by them that the procedure and constitution of the older societies should afford no precedent. They felt it undesirable that there should be a president; and that it would be conducive to greater interest in its welfare if a chairman were elected at each meeting. As few officials as possible should be elected, and such should consist of a council of twenty-four members, each holding office for not more than three consecutive years; a chairman of council, annually elected and not holding office for more than two years; and two honorary secretaries, whose appointments were terminable in three years, one resident in London to represent the London members and one in the provinces to personate the country members. In the following session, owing to the large amount of work, two honorary secretaries were appointed for London. Another permanent office, in addition to that of treasurer, was subsequently created, viz. the editor of the reports.

A further departure from precedent was the rule empowering the Society to hold at least one of its meetings, preferably in the summer, in one of the provincial centres, thus bringing London members in touch with their interested and enthusiastic *confrères* in the provinces.

At the inaugural meeting the following gentlemen were appointed to serve on the Council: Mr. Henry G. Armstrong, Dr. Henry Ashby, Dr. Fletcher Beach (West End Hospital for Nervous Diseases), Dr. George Carpenter (Evelina Hospital for Children), Dr. Edmund Cautley (Belgrave Hospital for Children), Dr. Wayland C. Chaffey, Dr. Arthur Downes, Mr. Walter Edmunds, Dr. Theodore Fisher,

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* The original rules, together with subsequent alterations, are contained in each volume of the 'Reports.'

Dr. Leonard Guthrie (Paddington Green Children's Hospital), Mr. Robert Jones (Liverpool Royal Southern Hospital), Dr. David B. Lees, Dr. Lewis Marshall, Dr. John McCaw, Mr. D'Arcy Power, Dr. Humphrey D. Rolleston, Dr. A. Ernest Sansom, Dr. George E. Shuttleworth, Mr. Harold J. Stiles (Edinburgh Hospital for Children), Dr. George A. Sutherland, Dr. James Taylor (North-Eastern Hospital for Children), Mr. Alfred H. Tubby, Mr. William H. Vicary (Newcastle Hospital for Children), and Dr. Dawson Williams.

Mr. R. Clement Lucas was appointed Honorary Treasurer, Dr. Charles H. Willey (Sheffield Children's Hospital) Provincial Secretary, and Mr. Sydney Stephenson London Secretary.

Dr. A. Ernest Sansom was unanimously elected Chairman of Council.

The original members were :

Mr. F. C. Abbott (Evelina Hospital).

Dr. B. L. Abrahams.

Dr. A. L. Achard.

Dr. A. J. Alliott (Sevenoaks).

Mr. H. G. Armstrong.

Dr. Henry Ashby (Manchester Hospital for Children).

Mr. Thomas J. Barnardo (Barnardo's Homes for Waifs and Strays).

Dr. Fletcher Beach (West End Hospital).

Dr. Walter Cardy Bluck (Southend).

Dr. Joseph Blumfeld (St. George's Hospital).

Mr. Albert Boyd-Wallis (Brentwood).

Mr. Frank Brightman (Broadstairs).

Dr. Hablot Browne (Hoylake).

Mr. F. F. Burghard (King's College Hospital and Paddington Green Children's Hospital).

Mr. C. G. Burton (Clapton).

Dr. James Carmichael (Edinburgh Hospital for Sick Children).

Dr. George Carpenter (Evelina and North-Eastern Hospitals for Children; subsequently Editor and

Founder of 'The British Journal of Children's Diseases' and Editor for eight years of the 'Reports' of the Society).

Mr. Herbert L. Carre-Smith.

Dr. Edmund Cautley (Belgrave Hospital for Children).

Dr. Wayland C. Chaffey (Royal Alexandra Hospital for Children, Brighton).

Dr. Charles W. Chapman (Hospital for Diseases of the Heart).

Dr. William Chubb (Sandgate).

Dr. Ernest Clarke (Central London Ophthalmic Hospital).

Mr. J. J. Clarke (City Orthopaedic Hospital).

Dr. W. E. A. Cummins (Cork Hospital for Women and Children).

Mr. Frank Deas (Wimbledon).

Mr. Clinton Dent (Belgrave Hospital for Children).

Dr. Morgan Dockrell (St. John's Hospital for Diseases of the Skin).

Dr. Arthur Downes (Local Government Board Medical Inspector).

Mr. Douglas Drew (North - Eastern Hospital for Children).

Mr. Walter Edmunds (Evelina Hospital for Children).

Dr. C. Elliott (Bristol Hospital for Women and Children).

Dr. William Ewart (St. George's Hospital and Belgrave Hospital for Children).

Dr. W. Soltau Fenwick (Evelina Hospital for Children).

Dr. Theodore Fisher (Bristol Hospital for Sick Children).

Dr. James K. Fowler (Middlesex Hospital).

Dr. T. Colcott Fox (The Westminster Hospital and Paddington Green Children's Hospital).

Mr. Andrew Fuller (Local Government Board Medical Inspector).

Mr. Henry Edmund Giffard (Egham).

Mr. Leonard Thomason Giles (Scarborough).

Dr. Clement Godson (City of London Lying-in Hospital).

Dr. A. C. Gray.

Dr. Leonard Guthrie (Paddington Green Children's Hospital).

Dr. Charles N. Gwynne (Sheffield Children's Hospital).

Mr. G. L. L. Hawken (Hurstpierpoint).

Dr. David Mount Hutton (Southport).

Dr. Henry Richmond Hutton (Manchester General Hospital for Children).

Mr. Francis Jaffrey (St. George's Hospital and Belgrave Hospital for Children).

Mr. W. H. H. Jessop (St. Bartholomew's Hospital).

Mr. Robert Jones (Royal Southern Hospital, Liverpool).

Mr. H. W. Kendal (Bristol Hospital for Children).

Mr. George Knapton (Manchester).

Dr. H. L. Lack (The London Hospital and Paddington Green Children's Hospital).

Dr. Sydney Lambert (Harrow-on-the-Hill).

Mr. Alfred Owen Lankester.

Dr. Arnold Lawson (The Middlesex Hospital and Paddington Green Children's Hospital).

Mr. Philip G. Lee (Women and Children's Hospital, Cork).

Dr. David B. Lees (St. Mary's Hospital and Great Ormond Street Children's Hospital).

Dr. Percy G. Lewis (Folkestone).

Dr. T. D. Lister.

Mr. R. Clement Lucas (Guy's Hospital and Evelina Hospital for Children).

Dr. John McCaw (Belfast Hospital for Children).

Dr. Dan McKenzie (Leytonstone).

Dr. R. G. McKerron (Children's Hospital, Aberdeen).

Mr. John Malcolm (Samaritan Free Hospital).

Mr. C. Devereux Marshall (Victoria Hospital for Children).

Dr. Lewis Marshall (Nottingham Children's Hospital).

Mr. C. H. Milburn (Victoria Hospital for Children, Hull).

Dr. Paul Moline (Victoria Hospital for Children).

Mr. Isaac Mossop (Bradford Children's Hospital).

Dr. Porter Parkinson (North-Eastern Hospital for Children).

Dr. Frank Penrose (Great Ormond Street Children's Hospital).

Dr. A. W. C. Peskett (Nottingham).

Mr. D'Arcy Power (St. Bartholomew's Hospital and Victoria Hospital for Children).

Dr. D. L. Roberts (Manchester Royal Infirmary).

Mr. H. B. Robinson (St. Thomas's Hospital and East London Hospital for Children).

Dr. Bertram H. Rogers (Bristol Hospital for Sick Children).

Dr. H. D. Rolleston (St. George's Hospital and Victoria Hospital for Children).

Mr. Bernard Roth (Royal Alexandra Hospital for Children, Brighton).

Mr. G. L. Rugg (Norwood).

Dr. A. Ernest Sansom (The London Hospital and North-Eastern Hospital for Children).

Dr. Henry Arthur Sansom.

Dr. James H. Sequeira (The London Hospital and North-Eastern Hospital for Children).

Dr. C. E. Shelly (Haileybury College, Hertford).

Dr. George E. Shuttleworth (Royal Asylum for Imbeciles, Lancaster).

Dr. Ernest Snape

Dr. M. C. Staunton (Children's Hospital, Dublin).

Dr. Hugh Stewart.

Mr. Sydney Stephenson (Evelina and North-Eastern Hospitals for Children ;, subsequently Editor and Founder of the 'Ophthalmoscope').

Mr. Harold J. Stiles (Royal Edinburgh Hospital for Sick Children).

Mr. T. H. Summerhill (Alexandra Children's Hospital, Rhyl).

Dr. Septimus Sunderland (Royal Hospital for Children and Women).

Dr. George A. Sutherland (Paddington Green Children's Hospital).

Dr. James Taylor (North-Eastern Hospital for Children).

Dr. StClair Thomson (Throat Hospital, Golden Square).

Mr. Alfred H. Tubby (The Westminster Hospital and Evelina Hospital for Children).

Mr. Francis Vacher (Wirrel Hospital for Children, Birkenhead).

Mr. W. H. Vicary (Children's Hospital, Newcastle-on Tyne).

Dr. Dawson Williams (East London Hospital for Children; Editor of the 'British Medical Journal').

Dr. Edward C. Williams (Bristol Hospital for Sick Children).

Dr. Charles H. Willey (Sheffield Children's Hospital).

Mr. J. H. Wilks (Sheffield Children's Hospital).

Dr. Kemlo Wilson (Hanwell).

The first meeting of the newly-formed Society was held at the rooms of the Medical Society, Chandos Street, W., on Friday, October the 19th, 1900, and cases were exhibited by Mr. R. Clement Lucas, Dr. James Taylor, Mr. A. H. Tubby, Dr. G. A. Sutherland, Mr. Sydney Stephenson, Dr. George Carpenter, Dr. J. H. Sequeira, and Dr. Theodore Fisher (Bristol).

Subsequent meetings of the Society took place at various children's hospitals, viz. Evelina Hospital (November, 1900), East London (January, 1901), Paddington Green (February), Belgrave (Gloucester Street, S.W.) (March), North-Eastern (April), and Victoria Hospital (May). The meetings were well attended, the average number present being about forty.

The first provincial meeting of the Society was held at Liverpool on Saturday, June the 29th, 1901, and the local arrangements were made by Mr. Robert Jones (Liverpool), who occupied the chair at the Medical Institution, Hope Street. Papers were read by Dr. George Carpenter and Mr. Sydney Stephenson, Dr. D. M. Hutton (Southport), Dr. Richard Caton (Liverpool) (introduced), Mr.

Keith Monsarrat (Liverpool), Dr. Charles J. Macalister (Liverpool), Dr. J. H. Bryant, Mr. R. Clement Lucas (by proxy), Dr. James Carmichael (Edinburgh) (by proxy), and Dr. John McCaw (Belfast) (by proxy).

Prior to the meeting a large number of interesting cases were shown by Mr. Robert Jones and others, and there was an attendance of seventy members and guests.

In the evening there was a dinner at the Adelphi Hotel, and Dr. A. Ernest Sansom took the chair.

The following day members were entertained at luncheon by Mr. Robert Jones, and subsequently crossed by the ferry to Birkenhead and were driven by him to the West Kirby Home for Convalescent Children. Here a ward containing twenty beds (opened in 1899) had been placed at the disposal of those who were responsible for the present Liverpool Country Hospital for Children—one of the pioneer hospitals for the open-air treatment of children's complaints. Subsequently the site selected for the Liverpool Country Hospital was viewed. There were present, among others, on this occasion Dr. A. Ernest Sansom, Mr. Walter Edmunds, Mr. Alfred H. Tubby, Dr. Theodore Fisher (Bristol), Dr. G. E. Shuttleworth, Mr. Sydney Stephenson, Dr. Edmund Cautley, Dr. George A. Sutherland, and Dr. J. H. Bryant, also Dr. J. H. Abram, Mr. Damer Harrisson, Mr. S. S. Taylor, Dr. Charles A. MacAlister, Dr. George P. Newbolt, Mr. Thelwall Thomas, and Dr. John R. Logan, all of Liverpool, and Mr. W. McAfee, of Birkenhead.

According to the rules of the Society the chairmen were elected for each meeting by the members present, and this custom was continued throughout its existence. During the first year of the Society's establishment the following members were nominated to the position, viz. Dr. A. E. Sansom (the first meeting), Mr. R. Clement Lucas (at the Evelina Hospital), Dr. Dawson Williams (at the East London Hospital), Mr., now Sir, Watson Cheyne, Bart. (at the Paddington Green Children's Hospital), Dr. Edmund Cautley (at the Belgrave Hospital), Dr. A. E.

Sansom (at the North-Eastern Hospital), and Mr. D'Arcy Power (at the Victoria Hospital).

On July the 26th, 1901, the Society held its first annual general meeting under the chairmanship of Dr. A. Ernest Sansom (Chairman of Council), at No. 11, Chandos Street, W.

At this meeting it was announced that during the session the membership had increased from 102 to 237. Of these, 144 belonged to London and its suburbs, 82 to the provinces, 4 to Scotland, and 7 to Ireland. The Treasurer's accounts were audited by Mr. Arnold Lawson (Paddington Green Hospital for Children) and Mr. Andrew Fuller (Medical Inspector Local Government Board), and there was a balance in favour of the Society of £166. 14*s.* 9*d.* The Council also reported that they had arranged for the issue of a volume of 'Reports' as soon as possible.

Dr. George Carpenter was appointed Medical Secretary for the ensuing session (1901-2), Dr. Theodore Fisher (Bristol) taking the place of Dr. Willey (Sheffield) as Provincial Secretary, and the reponsibility of giving effect to the Council's intention of producing 'Reports' was left entirely in Dr. Carpenter's hands. There has resulted from his gratuitous annual labours volumes I to VIII, the style and setting of which he designed, the type being specially cast for the work. These volumes contain, in chronological order, the doings of the Society from the hour of its foundation to the time of its voluntary dissolution and fusion with the Royal Society of Medicine in 1908. To those, therefore, who may feel desirous for greater detail, these volumes will afford full and accurate information. The Society is indebted to Mr. Robert Evan Adlard, the printer of these volumes, who has taken the keenest interest in its pioneer work in England, and who has ever been ready to give the Editor the benefit of his special knowledge in regard to the printing and to the production of the illustrations.

As will be gathered from a perusal of its history further on, the Society has always been threatened with the

Bogey of opposition, but the opposition has never been of a strenuous character. In its early days it is no secret that those inimical to the Society started in a spirit of self-proclaimed though not obvious superiority a rival body, but there is no record in literature of its labours.

During its second Session meetings were held alternately at children's hospitals, viz. Great Ormond Street, The Evelina, Paddington Green, and The North-Eastern Children's Hospitals, and at the Rooms of the Medical Society, Chandos Street.

The Provincial Meeting was held at the Pendlebury Hospital for Children, Manchester, Dr. Henry Ashby in the chair, and prior to the meeting various interesting cases were demonstrated in the wards by Dr. Ashby and the medical and surgical staff. Among those who attended were Mr. R. Clement Lucas, Dr. Theodore Fisher, Dr. H. R. Hutton (Manchester), Mr. Walter Edmunds, Dr. George Carpenter, Dr Edmund Cautley, Dr. A. Ernest Sansom, Dr. Charles MacAlister (Liverpool), Dr. George A. Sutherland, Dr. W. Page May, Dr. Joshua Cox (Manchester), Dr. C. N. Gwynne (Sheffield), Dr. C. H. Willey (Sheffield), Mr. Sydney Stephenson, and Mr. R. C. Dunn (Liverpool).

In the evening there was a dinner at the Queen's Hotel, Manchester, at which Dr. Henry Ashby presided. The arrangements for the provincial meeting were left in the hands of Dr. Henry Ashby, who, on the following day, Sunday, conducted a party which took train from Manchester to Grindelford, drove to the Peacock Inn, Baslow, for luncheon, visited Chatsworth, drove back to and took tea at Bakewell, and subsequently left by train from Rowsley.

The Annual General Meeting at the Hotel Cecil in 1902 was followed by a dinner, at which Dr. Henry Ashby, of Manchester, presided. It was announced that the Council had decided to hold a special meeting in December for the purpose of discussing tubercular peritonitis;

and, further, that they had also resolved that all the London meetings of the Society during the following Session (1902–1903) should be held at a central meeting-place. The original intention of the governing body was that all the children's hospitals should be visited in turn, but that idea had to be abandoned. The Society, when it had paid for its first volume of 'Reports,' was able to show a balance of £200. 15*s.* 10*d.*

During the Session of 1902–1903, Dr. A. Ernest Sansom, having served two years as Chairman of Council, was succeeded by Mr. Alfred H. Tubby, and Dr. George Carpenter succeeded Mr. Sydney Stephenson as Senior Secretary, the vacancy created being filled by Dr. George A. Sutherland.

In the first three years of the Society's existence all the clerical work was undertaken by the Secretaries, with the object of husbanding the Society's resources. The bulk of the work of the Society had necessarily to be undertaken by the London Secretaries, and the Provincial Secretaries were not able to afford any material aid. But in subsequent years its continued prosperity obviated any such additional labour and tax upon their leisure.

The services rendered to the Society by Mr. Sydney Stephenson during the first year cannot be allowed to be passed over without warm recognition. During this period the whole of the clerical work devolved upon him, and it would have been impossible to make a better selection for the responsible position he so ably occupied. His business-like methods, his energy, his determination to make the Society "go," and the vast amount of time he devoted to its interests, can only be thoroughly appreciated by those who were brought into intimate contact with him during this trying and critical period. He was not only one of its founders but also one of its chief supports and most regular attendant.

And it will not be inopportune to recall that the other partner in the idea of starting the Society, Mr. A. H. Tubby, promptly brushed aside the suggestion that he should be nominated as its first Chairman of Council, because he thought that the post should be held by an older man. He was equally self-denying in declining to allow his name to be brought forward as a candidate for the first Wightman Lecture. It will also be remembered that in 1903, at the Provincial Meeting at Brighton, the paper he read on "The Urban Hospital Treatment of Surgical Tuberculosis: Is it Justifiable?" drew attention to the outdoor treatment of surgical tuberculosis and the establishment of homes for that purpose.

The Provincial Meeting, which was very well attended, was held at Brighton on Saturday, June the 20th, 1903, at the Royal Alexandra Hospital for Sick Children, with Dr. Wayland C. Chaffey, of Brighton, in the chair. Prior to the meeting many cases of interest were shown by Dr. W. C. Chaffey, Dr. Edmund Hobhouse, and other members of the medical and surgical staff. There were also present, among others, Mr. R. Clement Lucas, Dr. A. Ernest Sansom, Mr. Alfred H. Tubby, Mr. Clinton Dent, Mr. Francis Jaffrey, Mr. R. H. Parry (Glasgow), Dr. Edmund Cautley, Dr. Theodore Fisher, Dr. George A. Sutherland, Mr. George Pernet, Dr. Alexander Morison, Dr. Eldon Pratt (Henfield), Mr. Sydney Stephenson, Mr. Walter Edmunds, Dr. G. E. Shuttleworth, Dr. Travers Smith, Dr. A. J. Alliott (Sevenoaks), Dr. W. Cardy Bluck (Southend), Dr. John Braithwaite (Buxton), Dr. Fletcher Beach, Dr. Robert Hutchison, Mr. F. F. Burghard, and Dr. Charles W. Chapman.

At the Annual General Meeting, held at 25, Weymouth Street, W., on July the 23rd, 1903, Mr. A. H. Tubby (Chairman of the Council) in the chair, the Council announced that owing to the liberality of Mr. Charles Wightman, and through the instrumentality of Mr. R. Clement Lucas, the former had generously offered the Society an annual donation of £20. to deliver a lecture, to be called the "Wightman Lecture," first, as a permanent memorial to his deceased son, Alexander Cockburn Wightman, aged 7 years, and, secondly, with the hope that this lecture might be the means of establishing or of diffusing such knowledge as would tend to lower the mortality of diseases to which children are liable. It may be mentioned that the boy died of diphtheria in the pre-antitoxin days, to the great grief of his father, whose only son he was, and that in spite of tracheotomy performed by Mr. R. Clement Lucas, and of the most skilled medical attendance.

Not only is the Society indebted to Mr. Clement Lucas for the Wightman Lecture, but it may be asserted without fear of contradiction that had it not been for him its history would have been a very different affair to that now recorded. By acclamation he was appointed Treasurer, the only permanent office then in the Society's gift, and how ably he has carried out his duties the flourishing state of the Society's finances and its large invested capital bear ample evidence. But it is not so much to his position as Treasurer as to that of one of the fathers of the Society that the Society is under so great an obligation to him. He spoke and fought valiantly for its welfare. He spared no effort to interest all—young and old alike—in the new body. He made the actively hostile into converts, he guided the hesitating footsteps of others, he procured many new members, he was a veritable tower of strength to his colleagues, and he gave ample demonstration that he rejoiced to be associated with younger men in such pioneer work. It is unfortunate that Mr. Clement Lucas's ideals for the Society's future could not be realised. The conception of a meeting house the property of the Society, the formation of a library, and the production of a pathological museum would have been of untold value to the welfare of children which he has so much at heart.

The Council decided that the Annual Dinner of the Society should be held at the first ordinary meeting in the month of October, 1903. This took place at the Hotel Cecil, on Friday, October the 16th, and was followed by the Annual Dinner. Dr. A. Ernest Sansom was in the chair, and fifty-five members and their guests were present.

On May the 6th, 1904, a special meeting was held, and Mr. Harold J. Stiles and Dr. Stuart McDonald (introduced), both of Edinburgh, read a paper on delayed chloroform poisoning.

The Provincial Meeting was held on Saturday, June the 18th, 1904, in the Medical Library, University College, Bristol, Dr. Theodore Fisher, of Bristol, being in the chair. Prior to that cases were shown at the Hospital for Sick Children, St. Michael's Hill, by Dr. Bertram H. Rogers, Dr. E. Cecil Williams, Dr. Fortescue-Brickdale, and Dr. Theodore Fisher. Surgical cases were exhibited by Mr. Charles A. Morton (introduced), Mr. H. W. Kendall (introduced), and Mr. H. Elwin Harris (introduced). The meeting was well attended, some fifty members and visitors being present, among them being Dr. Frederick Taylor, Mr. R. Clement Lucas, Mr. Alfred H. Tubby, Mr. George Pernet, Dr. James Taylor, Dr. George A. Sutherland, Dr. George Carpenter, Dr. Edmund Hobhouse (Brighton), Dr. Leonard Guthrie, Mr. Richard Lake, Dr. Walter Swayne (Bristol) (introduced), Dr. G. Markham Skerrit (Bristol) (introduced), Mr. C. A. Morton (Bristol) (introduced), Mr. J. Michell Clarke (Bristol) (introduced), Mr. J. Lacy Firth (Bristol) (introduced), and Mr. R. G. Poole Lansdown, (Bristol) (introduced).

The Provincial Dinner was held at the Clifton Down Hotel, Clifton, Dr. Frederick Taylor in the chair.

The first Wightman Lecture was delivered by Mr. R. Clement Lucas, at the Hotel Cecil, Strand, at the conclusion of the Annual General Meeting, on July the 15th, 1904, the subject being on "The Hereditary Bias and Early Environment in their Relation to the Diseases and Defects of Children."

The proceedings were followed by a dinner, Mr. Sydney Stephenson, the chairman of Council, who had succeeded Mr. Alfred H. Tubby in that office, in October, 1903, occupying the chair. Dr. Edward Cautley, during the session 1904–5, succeeded Dr. George Carpenter, who had held office as Secretary for three years, and Dr. Theodore Fisher, of Bristol, was succeeded by Mr. R. H. A. Whitelocke, of Oxford, as Provincial Secretary. On the expiration of Dr. Carpenter's term of office as Secretary he was desired by the Council to continue to edit the 'Reports,' and for that purpose an extra office was created, viz. "Honorary Editor of Reports," which Dr. Carpenter held to the end.

During the sessions of 1902–3 and 1903–4 the meetings of the Society were held at the rooms of the Medical Society, 11, Chandos Street, W., excepting the localities previously mentioned, and the chair was taken by various prominent London and provincial members. The Society continued to grow and flourish financially. On the date of the Annual General Meeting in 1904 it numbered 304 members, it had £228. 13*s*. 6*d*. invested in Consols at 2½ per cent., and a balance of £149. 9*s*. 10*d*. at its bankers.

During the Session of 1904–5 the total membership was 332, and 48 new names were added to the list. On December the 16th, 1904, a special meeting was held for the discussion of "Sudden and Unexpected Death in Children."

The Wightman Lecture was delivered by Dr. Henry Ashby (Manchester), on "Some of the Neuroses of Early Life," at the rooms of the Medical Society, Chandos Street, W.

The Provincial Meeting was held at Sheffield on Saturday, June the 17th, 1905. Cases were exhibited at the Children's Hospital prior to the General Meeting by Dr. C. N. Gwynne, Dr. C. H. Willey, Dr. J. H. Wilks, Mr. Simeon Snell (introduced), Dr. Thomas H. Morton (introduced), and Dr. Harold Leader (introduced), all of Sheffield, and specimens were shown by Professor Arthur Hall (introduced), Dr. J. H. Wilks and Dr. Harold Leader (introduced) of the same city, and by Dr. J. G. Emanuel (Birmingham). Dr. Willey occupied the chair during the reading of the papers.

Among those present were Mr. H. Betham Robinson, Dr. George A. Sutherland, Mr. R. Clement Lucas, Dr. Edmund Cautley, Mr. Sydney Stephenson, Mr. J. Jackson Clarke, Dr. George Carpenter, Mr. R. C. Dunn (Liverpool), Mr. Alfred H. Tubby, Mr. H. S. Clogg, and Dr. John Braithwaite (Buxton). In the evening the members and their friends dined together at the Royal Victoria Station Hotel, Dr. C. N. Gwynne being in the chair.

The following day a coach drive was taken to Baslow and back with luncheon at the Peacock Inn, the arrangements for which were made by Dr. C. N. Gwynne.

The Annual General Meeting took place at 11, Chandos Street, W., on Wednesday, July the 19th, 1905, and the Treasurer announced that the Society had £541. 10*s*. 2*d*. invested and a balance at their bankers of £206. 4*s*. 2*d*. All the meetings, with the exception of the Provincial, were held at No. 11, Chandos Street, W.

For the session 1905–6 there was a further change of officers. Mr. R. Clement Lucas succeeded Mr. Sydney Stephenson as Chairman of Council. Dr. George A. Sutherland, after holding office for two years, resigned and was succeeded as Senior Secretary by Dr. Edmund Cautley, Mr. Francis Jaffrey being elected to fill the vacancy created.

A special meeting was held on December the 15th, 1905, for a discussion on "Pleural Effusions, Serous and Purulent."

On Friday, May the 4th, a meeting was devoted to the exhibition of pathological specimens.

The Wightman Lecture was delivered on May the 18th, 1906, by M. le Docteur Broca, Surgeon to the Hôpital des Enfantes Malades, Paris, and President of the Société de Pédiatrie of Paris, on "Appendicitis, Acute and Chronic." M. Broca was subsequently entertained by the Society at a dinner at the Imperial Restaurant, Regent Street, W., at which about one hundred members and their guests were present. On this occasion Sir William Broadbent, Sir Thomas Barlow, and other distinguished members of the medical profession, were the guests of the Society.

The Provincial Meeting of 1906 was held at the Radcliffe Infirmary, Oxford, on Saturday, June the 23rd. Prior to the general meeting cases and specimens were exhibited by Mr. A. P. Parker (Oxford) (introduced), Mr. H. P. Symonds (Oxford) (introduced), Mr. Ernest Mallam (Oxford) (introduced), Mr. P. H. Adams (Oxford) (introduced), Mr. George Pernet, Dr. W. J. Turrell (Oxford) (introduced), Mr. R. H. A. Whitelocke, Professor James Ritchie (Oxford) (introduced), Professor Thompson (Oxford) (introduced), and Mr. Finch (Oxford) (introduced). Dr. William Osler (Oxford) (introduced), the Regius Professor of Medicine, occupied the chair at this stage of the proceedings, and at the meeting proper Dr. William Collier (Oxford) (introduced) was the chairman.

The local arrangements were made by Mr. R. H. A. Whitelocke, of Oxford, the Provincial Secretary. A large number of local medical men were present as the guests of the Society, and there were also present Mr. Alfred H. Tubby, Mr. R. Clement Lucas, Dr. Bertram H. Rogers (Bristol), Dr. Leonard Guthrie, Dr. Percy Lewis (Folkestone), Dr. George A. Sutherland, Dr. Edmund Cautley, Mr. Howell Evans, Dr. Charles W. Chapman, Dr. E. C. Williams (Bristol), Mr. Sydney Stephenson, and Dr. George Carpenter.

The Annual General Meeting was held on July the 20th, 1906, and the Council gave their report, after most careful consideration, and arrived at after numerous meetings, on the proposed amalgamation of the chief medical societies of London into one large society, to be called the Royal Society of Medicine. The Council were by no means unanimous that amalgamation was in the best interests of the profession, or that the financial basis of the scheme was satisfactory. Further, it did not appear that any provision had been made to safeguard the position of the Society as a permanently independent section and the continuance of its annual publication of a volume of 'Reports.' In order to ascertain the views of the members a postcard circular was sent out inviting an opinion on the question. In this circular the reasons which influenced the Council in forming their opinion were not formulated. The result showed that a preponderance of country members were opposed to amalgamation, viz. 43 against 34, and a preponderance of London members were in favour of it, viz. 76 against 47.

Of the then existing 28 Members of Council, 18 were opposed to amalgamation, 4 were in favour of it, and 6 did not record any opinion.

The balance-sheet showed £500. invested in Consolidated Stock 2½ per cent., with a balance at the bankers of £594. 3*s*. 10*d*.

Dr. Edmund Cautley having resigned the office of Secretary after two years' service, Mr. Francis Jaffrey succeeded him as Senior Secretary, and Dr. Porter Parkinson was elected to fill the vacancy during the session of 1906–7. Mr. Henry Skelding, of Bedford, succeeded Mr. R. H. A. Whitelock, of Oxford, who had held office for two years as Provincial Secretary.

During the Session of 1906–7 a special meeting was held at No. 11, Chandos Street, W., on Friday, November the 23rd, 1906, to receive the following resolution recommended for adoption by the Council: "That The Society for the Study of Disease in Children declines under present circumstances to join in the scheme of amalgamation." Mr. R. Clement Lucas, the Chairman of Council, occupied the chair. Mr. Lucas acted as the representative of the Council of the Society during the preliminary investigations which were undertaken to promote the formation of a Royal Society of Medicine by the amalgamation of a number of the London medical societies, and the idea of which took form at a meeting at the College of Physicians in the summer of 1905. He held a watching brief with instructions not to commit the Society in any way. This meeting is reported in full in vol. vii of the 'Reports,' pp. xiv–xxxvii, under the heading "Union of Medical Societies of London." Dr. C. O. Hawthorne (Member of Council), Dr. George A. Sutherland

(Member of Council), Dr. Frederick Taylor, Mr. Lockhart Mummery, and Dr. Leonard Guthrie spoke in favour of the scheme, and Mr. R. Clement Lucas (Chairman of Council), Dr. W. C. Chaffey (Brighton), Mr. Sydney Stephenson, Mr. H. L. Carre-Smith, Mr. Milner Burgess (Harlesden), and Mr. George Pernet against it. The following objections were advanced against amalgamation, viz. that the scheme was financially unsound; that the 'Reports' in their present form would cease; that as a section there would be little incentive to obtain new members; that the Society would lose its accumulated capital, its name, and its independence with its possibilities of future progress; that there was no guarantee that the Children's Section would always remain distinct; that the incentive to the study of children's diseases in this country would again lapse, and that for this great sacrifice it would obtain nothing in return. For the scheme it was advanced that the advantage which would result to the profession from a proper scheme of amalgamation were very considerable; that if the Society did not amalgamate there would undoubtedly be formed a section for the study of children's diseases; that those who were acquainted with the genesis of the Society would recognise that there were certain medical men who for good reasons or otherwise would take a leading part in the creation of such a section; that in such an event the attractions the new Society could offer in the way of value for money were very considerable, and that in consequence defections would certainly follow. The pessimistic views as to finance were also controverted, and it was advocated that there was some advantage in the Society contributing to a large concern which would be representative of the whole of medicine and surgery in all its departments, not only in London but in the United Kingdom. As a result of that meeting there voted in favour of the resolution 22 and against it 5.

A special meeting for the discussion of "Rickets" was held on Friday, December the 14th, 1906, and the

Wightman Lecture on "Pneumonia in Children" was delivered by Dr. Frederick Taylor on Friday, May the 17th, 1907.

The Provincial Meeting was held at the County Hospital, Bedford, on Saturday, June the 20th, 1907, the local arrangements being in charge of Mr. Henry Skelding.

Prior to the meeting proper, cases and morbid and other specimens were shown at the County Hospital by Dr. Harvey Goldsmith (Bedford), Mr. Henry Skelding (Bedford), Mr. S. J. Ross (Bedford) (introduced), Mr. C. G. Johnson (Bedford) (introduced), Mr. W. A. Sharpin (Bedford) (introduced), Dr. Willmer Phillips (Bedford) (introduced), Mr. W. Gifford Nash (Bedford), Dr. H. W. L. Waller (Bedford) (introduced), and by Mr. G. T. Verry (Bedford) (introduced). At the meeting, which also took place at the County Hospital, the chair was taken by Dr. R. H. Coombs (Bedford) (introduced).

Among others there were present: Mr. Francis Jaffrey, Dr. J. Porter Parkinson, Mr. R. Clement Lucas, Dr. F. W. Higgs, Dr. Edmund Cautley, Mr. Alfred H. Tubby, Dr. Septimus Sunderland, Dr. George Carpenter, Dr. Percy Lewis (Folkestone), Mr. George Pernet, Dr. John Braithwaite (Buxton), Mr. Sydney Stephenson, Dr. William Hill, Dr. Alexander Morison, Dr. George A. Sunderland, Dr. Leonard Guthrie, Dr. William Ewart, Dr. C. O. Hawthorne, Mr. Milner Burgess (Harlesden), and Dr. Charles W. Chapman. In the evening there was a dinner at the Swan Hotel, with Mr. R. Clement Lucas in the chair.

At the Annual General Meeting, held on July the 19th, 1907, the treasurer announced that the expenses in regard to amalgamation amounted to £36. 5*s.* 8*d.* The invested capital of the Society amounted to £700. Consols, and that there was in hand a sum of £135. 12*s.* 8*d.*

For the session of 1907–8 there was a further change of officials. Dr. George Carpenter succeeded Mr. R. Clement Lucas as Chairman of the Council, who had held that office for two years. Dr. Porter Parkinson succeeded

Mr. Francis Jaffrey as Senior Secretary, and Mr. Hugh Lett was elected to fill the vacancy. Dr. J. G. Emanuel, of Birmingham, was appointed Provincial Secretary, vice Mr. Henry Skelding (Bedford).

The meetings of the Society, as heretofore, still continued to be held at the rooms of the Medical Society, 11, Chandos Street, W., at 5 p.m., half an hour earlier than during the first years of the Society's existence—viz. up to the end of the session 1905–6. The custom was still maintained of electing a chairman to preside at the meetings from among the members present, and the position was often filled by the country members of repute.

On Friday, December the 13th, 1907, a special meeting was held for the discussion of "Inherited Syphilis."

On Friday, May the 22nd, 1908, Sir W. Watson Cheyne, Bart., F.R.S., delivered the Wightman Lecture on "The Defensive Arrangements of the Body as illustrated by the Incidence of Disease in Children and Adults."

On Saturday, June the 20th, 1908, the Provincial Meeting was held at Birmingham. Dr. J. E. H. Sawyer (Birmingham) was appointed Additional Provincial Secretary in the unavoidable absence of Dr. J. G. Emanuel, and the local arrangements were left entirely in his hands. He worked so well and his management was so successful that over two hundred medical men took part, and this, the last Provincial gathering, proved to be the best attended. Prior to the meeting proper, cases and pathological specimens were exhibited in the Out-Patient Hall of the General Hospital. The members and guests were received by Sir Thomas Pye Chavasse, Senior Surgeon to the Hospital, and by Dr. George Carpenter, Chairman of Council. Cases and pathological and microscopical specimens were shown by Mr. Frank Barnes, Mr. Billington, Mr. Jameson Evans, Dr. Foxwell, Dr. Douglas Heath, Dr. Kaufmann, Mr. A. Lucas, Mr. Victor Milward, Mr. Nuthall, Dr. Stanley, Dr. Stacey Wilson, Dr. Stanley Barnes, Dr. Hewetson, Dr. James Miller, Dr. Wynn, and Dr. N. C. Penrose, all of Birmingham, and all introduced. Dr. James H. Sawyer

(Birmingham), Dr. Potts (Birmingham), and Dr. Walter Jordan (Birmingham), also exhibited cases and specimens.

The meeting for the reading and discussion of papers was held in the Out-patient Hall of the Children's Hospital, Dr. George Carpenter in the Chair. The members and their guests subsequently dined at the Grand Hotel, Sir James Sawyer, M.D., presiding.

The Annual General Meeting was held at No. 11, Chandos St., W., Dr. George Carpenter, Chairman of Council, presiding. At this Meeting Mr. Hugh Lett was elected to succeed Dr. Porter Parkinson as Senior Secretary to the Section for the Study of Disease in Children of the Royal Society of Medicine for the session commencing October, 1908, Dr. E. I. Spriggs being selected to fill the vacancy of Junior Secretary. Mr. Harold J. Stiles (Edinburgh) was elected to the post of provincial secretary. The Council announced that Dr. George Carpenter had been chosen Wightman Lecturer, and that the provincial meeting in 1909 would take place in Edinburgh.

It was announced that although a certain number of members had resigned owing to the Society not yet having joined the amalgamation of medical societies, yet so many new members had been elected that the roll had slightly increased. The actual number of the Society's members in August, 1908, was 310.

At a meeting of the Council on July the 8th, 1908, it was recommended, on the strength of the proposals made to the Society on behalf of the Royal Society of Medicine by their secretary, that the Society join that Society as the Section for the Study of Disease in Children. The terms agreed upon were that the Section would preserve its name "For the Study of Disease in Children." That all its members would be entitled to join the Royal Society of Medicine as fellows or members without entrance fee. That the Chairman of Council or his representative would represent the Society on the General Council of the Royal Society of Medicine. That the provincial meetings would be carried on as heretofore. That the Section would have absolute power as to the publication of the material brought before it. That members would be entitled to certain options in regard to the 'Transactions,' and that previous to amalgamation all expenses of the Society incurred during the session (1907–8) were to be discharged, including that of the volume of 'Reports' then in hand.

There were present Dr. Percy Lewis (Folkestone), Dr. Frederick Taylor (Trustee), Dr. Porter Parkinson (Secretary), Dr. F. W. Higgs, Dr. Leonard Guthrie, Mr. J. Scott Battams, Dr. E. I. Spriggs, Dr. Edward J. Nix, Dr. L. A. Clutterbuck, Dr. Charles W. Chapman, Mr. Russell Howard, Dr. C. O. Hawthorne (member of Council), Mr. Hugh Lett (Secretary), Mr. Sidney Gilford (Reading), Dr. T. R. Whipham (member of Council), Dr. William Ewart, Dr. G. L. Turnbull, Dr. George Pernet, and Dr. Joseph B. Wallace (Clapham).

The Treasurer's report was presented, which showed a total invested capital of £800. Consols, and that, after discharging all debts, there remained a balance at the bank of £100. 15*s*. 1*d*.

Mr. R. CLEMENT LUCAS said that in regard to certain hitherto contentious matters the Royal Society of Medicine were prepared to meet the Society, and that in the past it would have been gambling to risk such a strong financial position on unsecurity, but that now the Royal Society of Medicine was firmly established that objection no longer operated. He said what has been done cannot be undone, and the Society has compelled the recognition of the special study of disease in children in this country. Taking the whole matter into consideration, he thought the Society would now be wise in joining this amalgamation, because it was not desirable to divide those who are interested in children's diseases—we want, he said, to heal any dispute or discontent. The object of the Society, he maintained, is one, and a simple one, and that is, to do our very best in the interests of the study of disease in children.

Dr. C. O. HAWTHORNE drew attention to the fact that on the last occasion he was a member of a small and feeble minority, but that now, by a curious turn of affairs, they were in a position of happy unanimity.

Dr. George Pernet said he should certainly support the resolution, because it was joining on very different terms to those first offered; that now, instead of being whipped in like children, the Society was marching in with drums beating and colours flying.

Dr. William Ewart lamented the loss of £800., which would pass into the coffers of the Royal Society of Medicine, and for which no equivalent was offered; that as an independent society with some financial support the Society held a prouder and a better position, certainly a more unique position, than it would occupy when amalgamated. He said that several members a year ago, when this question was discussed, thought the Society possessed an asset which should not be sacrificed lightly, viz. its independence and energy, with perfect freedom for any developments which it might choose to undertake. In the future the Society would have no real action; it would be under orders. He did not think he could conscientiously vote for the expunging of the Society, although he was sure it would do admirable work and would enjoy many advantages which it could not have otherwise by joining the Royal Society of Medicine.

Dr. Frederick Taylor reminded Dr. Ewart that the function of the Society was not to collect money, but to promote the study of disease in children.

Dr. Leonard Guthrie predicted that the death of the Society would speedily take place if it declined to amalgamate, and he noted with extreme satisfaction that amalgamation was advocated by the Council.

The Chairman (Dr. George Carpenter) then put from the chair the following resolutions:

(1) That subject to the conditions circulated (*vide* p. xix) the Society agrees to the scheme of amalgamation.

(2) That the standing Committee (the present Chairman of Council, the Treasurer and the Secretaries), shall

be authorised to bring out the 'Reports' and to close the financial business of the Society previous to amalgamation.

(3) That a Special General Meeting be called to dissolve the Society subject to the conditions being fulfilled by the Royal Society of Medicine.

These resolutions were carried *nem con.*

Such, then, is the history in brief of The Society for the Study of Disease in Children from start to finish, and it cannot be denied that it has been a scientific and a financial success and also a powerful and a much-needed lever to compel the study of children's diseases in this country. That it did not receive, in its early days, support from a certain section of the medical profession presumably specially interested in children's complaints that ought to have been accorded to the undertaking on its merits cannot be laid to the door of the Society. The council were ever ready to welcome the co-operation of all men specially interested in the study of children's diseases in Great Britain—every inducement was held out to them to bear their share of the burden. The Society, whose sole object has been the interests of the children, has adopted amalgamation in the hope that the study of children's diseases in this country will gain an added impetus and an accession of force which will further promote that much-needed object.

The scientific work that has been accomplished by the Society in its short existence will be found within the covers of its eight volumes which will remain for all time a record of its labours and activity.

These eight volumes contain 384 pages of ordinary matter, 2760 pages recording the scientific work of the Society, together with 209 illustrations in the text, 39 plates, and 12 coloured plates. The small volume, No. IX, containing 104 pages devoted to a general index to the whole series, renders the work complete. The cost of printing and production amounted to about £1400.

An impartial critic will not, in the face of these facts, endorse the opinion that was once freely expressed by those who were hostile to it in the initial stages of the Society's work that "it had got into the wrong hands." The Society triumphantly refuted that ill-natured sneer; it has played the better and the more manly part, and its former members will in the future be able to look back upon its life and work with pride and with pleasure. Although the finger of Time may relentlessly point to the fact that some of the deductions and conclusions arrived at within its volumes have become old-fashioned and out of date, yet the spirit of progress which led to their birth will never be behind the times. And disciples of the art of healing in the future will gather that we too had our trials as well as our triumphs. They will understand that once living men like themselves, we too have passed across this world's stage, and that though our bodies are dust yet our spirits still speak to them in these pages, encouraging them to persevere and not to be daunted when the cause is noble, and to regard opposition and difficulties of research as a stimulus to increased effort. And it may be asked, Why was the Society such a success? And the reply to that is that all those who were actively interested in its welfare and in the subjects with which it dealt worked at it with heart and soul and missed no opportunities, individually and collectively, which would promote its advancement. All have lent a hand in the fashioning of the finished product exemplified by vol. viii of the 'Reports' with its index for the eight volumes under a separate cover, and their names will be found in the pages devoted to its history. But the work of a layman who has helped to make these 'Reports' must not be forgotten, that of the shorthand writer, Mr. H. Dickinson, who has so ably reported the Society's meetings, both London and Provincial, since its foundation.

During the eight years of its existence the Society has lost a number of its 102 original members, most by resignation, a few by death. The resignations numbered 34, viz. Mr. F. C. Abbott, Mr. H. G. Armstrong

PLATE I.

Dr. Charles Nelson Gwynne.
(Sheffield.)

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(Wellington College), Dr. J. Blumfeld, Mr. Boyd-Wallis (Brentwood), Dr. William Crabb (Sandgate), Dr. Ernest Clarke, Mr. J. Jackson Clarke, Dr. W. E. A. Cummins (Cork), Dr. Morgan Dockrell, Dr. Arthur Downs, Dr. C. Elliott (Bristol), Dr. W. Soltau Fenwick, Dr. James K. Fowler, Mr. L. T. Giles (Scarborough), Dr. Clement Godson, Mr. Robert Jones (Liverpool), Mr. H. W. Kendal (Bristol), Dr. H. L. Lack, Mr. Alfred Owen Lancaster, Dr. David B. Lees, Dr. F. D. Lister, Mr. C. D. Marshall, Mr. D'Arcy Power, Dr. D. L. Roberts (Manchester), Dr. H. D. Rolleston, Dr. Henry Arthur Sansom, Dr. James H. Sequeira, Dr. E. C. Shelley (Haileybury College), Dr. Ernest Snape, Dr. M. C. Staunton (Dublin), Mr. T. H. Summerhill (Rhyl), Dr. StClair Thomson, Mr. Francis Vacher (Birkenhead), and Mr. W. H. Vicary (Newcastle-on-Tyne). Five have unfortunately joined the majority, three of whom the Society could ill afford to lose.

The first to be called away was Charles Nelson Gwynne, Surgeon at the Children's Hospital, Sheffield, two years after the successful Provincial Meeting held in that city in the summer of 1904, and for which he was in part responsible. The son of Canon Gwynne, of Dublin, a descendant of the Knights of Gwynne, he was educated in Dublin and obtained the degrees of M.A. and M.D. at Trinity College.

A man of splendid physique and gentle manners, Dr. Gwynne easily won the love and confidence of the suffering and the young. "You could tell Dr. Gwynne anything" one of his patients recently said of him. Gwynne was always polite, but there was no weakness underlying it, he was as strong as steel, and nothing would keep him from the course he deemed to be the right one. He was a most sincere friend, always the same. If he could not say good of a man he spoke no harm. In all his actions he was most minutely painstaking, and his tenacity and perseverance over the most intractable cases were a noticeable trait.

For twenty-seven years his genial presence brought hope and cheerfulness into the wards of the Sheffield Children's Hospital, and he often led the Board of Management with his friendly counsel and wisdom. There and in his large private practice he was "a man greatly beloved."

Happy in his social and domestic relations, of literary tastes, and with a keen zest for life, Dr. Gwynne's Celtic light-heartedness and sense of humour made him an ideal comrade. Yet he gave his best to his profession. Everything else came second. Devoted to his work, he fell in harness, and died of "anæmia," at the age of fifty-nine.

It was but the other day at the Provincial Dinner at Birmingham that the Society learnt that Ashby of Manchester, Ashby of the Manchester Children's Hospital at Pendlebury, an institution whose name he had made world-famed, was ill. A telegram of sympathy met with a characteristic reply: "It is a great pleasure," so he wrote, "to think that one is not forgotten by one's friends when one is laid by." But it was not to be, and now not only Manchester but we all mourn his loss at the comparatively early age of 58.

Henry Ashby, Doctor of Medicine of the University of London, a man of strong personality—fine Henry Ashby; not only was he fine in intellect, but fine in physique, for he stood six feet in his stockings, and was broad in proportion. No "stove-pipe" hat encircled his brow, or frock coat—the modern conventional doctor's get-up—marred his comfort. Clad in a blue yachting suit or a cut-away coat in rough iron-grey cloth, with powerful, friendly, sun-tanned, freckled hands, the boon companions of sun and the salt sea, he was the very picture of unconventionality. No tight boots imprisoned his strong feet; a simple, crucial incision by his own hand through the leather of his boot over a bunion displayed his common-sense methods and his total disregard for the puerile criticisms of his neighbours. There was nothing

PLATE II.

DR. HENRY ASHBY.
(Manchester.)

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PLATE III.

DR ARTHUR ERNEST SANSOM.

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narrow-minded, or mean-spirited, or petty about Ashby, and no medical jealousies seared his soul or warped his judgment; he was as generous in his ideas as he was free in his dress. And his little patients, who, like dogs, instinctively recognise those who are fond of them and in sympathy with them, at once trusted him, and the tiny hand was not slow to be stretched out and grasped by the equally responsive, strong, friendly palm of Henry Ashby. His sympathy with children and his exquisite gentleness in handling them enabled him to make with ease the thorough examination which was his invariable custom. There was with Henry Ashby a deliberation and often a quaintness of expression in his speech which compelled attention, and at once excited interest in this dear, good fellow. Some men impress their day and generation by their work, others by their personality. Henry Ashby did both. Farewell, Henry Ashby; you have done noble work, and the Society could ill afford to lose you. But stay a moment; there is one benediction which we know would meet with your warm approval, and that is that your son, now a Resident at the hospital you loved and served so well, should worthily follow in your footsteps.

And last, but not least, the Society deplores the loss of Arthur Ernest Sansom, who was called away in 1907 at the age of 67. Future students of medical literature may feel desirous of knowing what manner of man was Sansom. The current medical papers have fully dealt with his medical career and with his work on the heart, but Sansom not only wrote about hearts medically, he was himself a man of heart, and he won hearts. When he associated himself with the Society he was in the late autumn of his career. Neat and quiet in his attire, gentle mannered, and of frail-looking physique, he literally brimmed over with geniality and goodness of heart. He was courteous to all, and warm in regard for all his friends. His many acts of charity were performed without ostentation and were known to few. Ever urged on to higher and nobler deeds, and inspired largely by his favourite poet Pope, he always held in view his favourite motto, "Learn to labour and to wait." Nothing appeared to put him out, he was urbanity itself, and he allowed nothing and nobody to put him out of love with the Society, its aspirations, and its future, and at the back of it all was the intense desire for the medical welfare of English children. That was his sole aim and object in taking a prominent position in The Society for the Study of Disease in Children, and he saw in the nascent Society a powerful lever to ensure the object which he had at heart and to remove the reproach under which English medicine laboured in regard to its incomprehensible disregard for the study of children's diseases.

In that determination he never wavered in spite of numerous powerful influences which were brought to bear upon him to induce him to dissociate himself from the newly-born venture. Sansom took a leading part in its initiation, he helped to draw out its rules, he presided at the inaugural meetings, and he was rightfully and appropriately elected its first Chairman of Council. He held that office for two consecutive years, and during that period by his tact and geniality, by his ripe experience, and by his great hospitality, he did much to knit together the forces of the Society and to provide a solid foundation on which to erect the present superstructure. Whether it was to take the chair at an ordinary meeting, or to preside over its social functions, Sansom was always ready to assist should his help be thought to be necessary. It seems but the other day that Sansom was the life and soul of the party of members that attended the first Provincial Meeting at Liverpool, and that one journeyed from Euston with him on that bright Saturday—bright as to the day, and bright as to the hope for the Society's future.

On the following day at the coach drive to West Kirby he was busy with his "kodak" "snapshotting" everything and everybody of interest, and his genial personality was diffused all around.

And now, alas, he too has joined the majority, and those of us who had the privilege to be associated with him will not forget him or his good example. Sansom gained a medical reputation for diseases of the heart, but the thing that will keep Sansom's memory green is the good work he did in helping to lay the foundation for the systematic study of diseases of children in England.

And now my work is accomplished, and before vacating the editorial chair of The Society for the Study of Disease in Children I cannot help feeling oppressed by mingled emotions—of thankfulness that my task is done, for it has been a strenuous one; of sorrowfulness that it should so soon be ended.

The Society has navigated its argosy past many a shoal, it has stoutly weathered with it many a storm, and it has safely anchored it in a friendly and a desirous port.

Its launch in 1900 and its historic voyage will prove as important to British children as was to the continent of America the sailing of the "Mayflower" in December, 1620, from Plymouth to its New England home.

From this stage it is foreseen that those who take this good work in hand will succeed in making the Study of Diseases in Children one of the most important departments in medicine, as well as the most attractive. I hold no mandate from the Society, but I am well aware that it is the heartfelt wish of every member that the Section for the Study of Disease in Children of the Royal Society of Medicine will do credit to its progenitor, and that it will also be endowed with its progenitor's energy, enterprise, and *bonne camaraderie*, and excel it in prosperity and production. We all wish it God-speed in its noble work.

GEORGE CARPENTER.

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BALANCE SHEET FOR THE YEAR ENDING JULY, 1908.

RECEIPTS.		£	s.	d.	EXPENDITURE.	£	s.	d.
To Balance brought forward		135	12	8	By Adlard & Son	145	6	0
,, Subscriptions		258	6	0	,, Hollebone Bros. & Trench (£100 Consols)	84	6	3
,, Dividends, Consols		19	0	0	,, G. Pulman & Sons	24	5	6
,, Messrs. Churchill, Sale of Reports		5	2	1	,, H. Dickinson	20	5	0
					,, The Medical Society	18	18	0
					,, G. Bethel	5	12	6
					,, Dr. Carpenter	5	0	0
					,, Dr. Skelding	3	14	0
					,, Dr. James E. H. Sawyer	2	19	11
					,, Dr. Porter Parkinson	2	0	0
					,, Mr. Hugh Lett	1	6	0
					,, Mr. W. H. Harris	0	10	0
					,, Treasurer for Petty Cash	3	0	0
					,, Cheque Book	0	2	6
Balance forward	100 15 1				,, Balance carried forward	100	15	1
		£418	0	9		£418	0	9

INVESTMENT.

Consolidated Stock, 2½ per cent. £800

WIGHTMAN LECTURE FUND.

		£	s.	d.		£	s.	d.
To Balance brought forward		£20	0	0	By Sir W. Watson Cheyne, Bart., C.B.	£20	0	0
,, Per Mr. Wightman		20	0	0				
Balance forward	£20 0 0				,, Balance carried forward	20	0	0
		£40	0	0		£40	0	0

We, the undersigned, have examined the foregoing accounts, together with the Vouchers and Bank Book, and find them to be correct

R. CLEMENT LUCAS, *Hon. Treasurer.*
July 14th, 1908.

CHAS. W. CHAPMAN.
E. I. SPRIGGS.

Meetings of the Society were held during the Session of 1907–1908 *at the undermentioned places.*

1. Friday, October the 18th, 1907.—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.
2. Friday, November the 15th, 1907.—No. 11, Chandos Street, Cavendish Square, W. Dr. George Carpenter in the Chair.
3. Friday, December the 13th, 1907 (Special Meeting for the discussion of "Inherited Syphilis"). No. 11, Chandos Street, Cavendish Square, W. Dr. G. E. Shuttleworth in the Chair.
4. Friday, January the 17th, 1908.—No. 11, Chandos Street, Cavendish Square, W. Mr. Sydney Stephenson in the Chair.
5. Friday, February the 21st, 1908.—No. 11, Chandos Street, Cavendish Square, W. Dr. Edmund Cautley in the Chair
6. Friday, March the 20th, 1908.—No. 11, Chandos Street, Cavendish Square, W. Dr. Charles W. Chapman in the Chair.
7. Friday, April the 10th, 1908.—No. 11, Chandos Street, Cavendish Square, W. Dr. Edmund Hobhouse (Brighton) in the Chair.
8. Friday, May the 8th, 1908.—No. 11, Chandos Street, Cavendish Square, W. Dr. William Ewart in the Chair.
9. Friday, May the 22nd, 1908 (the Wightman Lecture "The Defensive Arrangements of the Body as illustrated by the Incidence of Disease in Children and Adults," by Sir W. Watson Cheyne, Bart., F.R.S.).—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.
10. Saturday, June the 20th, 1908 (Provincial Meeting, Birmingham).—The General Hospital and at the Children's Hospital. Official Reception at the former by Sir Thomas Chavasse (introduced), Senior Surgeon, and by the Chairman of Council (Dr. George Carpenter). At the Children's Hospital the Chair was taken by the Chairman of Council.
11. Friday, July the 17th, 1908 (Annual General Meeting).—No. 11, Chandos Street, Cavendish Square, W. Dr. George Carpenter in the Chair.

LIST OF OFFICE BEARERS OF THE SECTION FOR THE STUDY OF DISEASE IN CHILDREN OF THE ROYAL SOCIETY OF MEDICINE, PAST MEMBERS OF COUNCIL AND OFFICE BEARERS OF THE SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN, WITH DATES OF SERVICE, AND MEMBERS OF THE SECTION FOR THE STUDY OF DISEASE IN CHILDREN OF THE ROYAL SOCIETY OF MEDICINE FOR THE SESSION 1908—1909.

(*Revised August*, 1908.)

(O.M. = Original Member. T. = Treasurer. TT. = Trustee.* S. = Secretary. S with date attached = Past Secretary. P.S. = Provincial Secretary. P.S. with date attached = Past Provincial Secretary. Ed. = Editor of the Reports. C. = Member of the Council. C. with date attached = Past Member of Council. * = Life Member. C.C. = Chairman of Council. C.C. with date attached = Past Chairman of Council. W. = Wightman Lecturer. W. with date attached = Past Wightman Lecturer.

By the amalgamation of the Society for the Study of Disease in Children with the Royal Society of Medicine the offices of Treasurer, Editor of the Reports, and Trustees were abolished.

Elected

O.M. Achard, Alexander Louis, M.D., 34a, Gloucester Place, W.

O.M. Alliott, Alexander John, M.D., Surgeon at The Hip Hospital, Sevenoaks; The Vine, Sevenoaks. (C. 1906–7, 1907–8.) (C.)

1907 Amy, A. C., M.B., Kensington General Hospital, Earl's Court, S.W.

1904 Anderson, C. M., M.D., 1, Elm Park Gardens, South Kensington, S.W.

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* Trustees were first appointed at the Annual General Meeting of 1907.

Elected

1903 Anderson, J. Ford, M.D., 11, Belsize Park, N.W. (C. 1903–4, 1904–5.)

1901 Archdale, Mervyn A., M.B., East Riding Lunatic Asylum, Beverley

1902 Armour, Donald John, M.B., Assistant-Surgeon at The West-London Hospital; Surgeon at The Belgrave Hospital for Children; 89, Harley Street, W.

1901 Armstrong, John T., Selby House, Beaumont Terrace, West Kensington, W.

1908 Atkinson, G. J., The Infirmary, New Cross, Wolverhampton.

1906 Bailey, B. E. G., Midhurst, Sussex.

1904 Batchelor, Charles, 1, Fairfield Avenue, Staines, Middlesex.

1901 Battams, J. Scott, 32, Cedars Road, Clapham Common, S.W.

1904 Baumann, E. P., M.D., Box 1257, Johannesburg, Transvaal, S. Africa.

O.M. Beach, Fletcher, M.B., Physician at The West-End Hospital for Diseases of the Nervous System; Chandos Road, Alton, Hants. (C. 1900–1, 1901–2, 1902–3.)

1906 Beatty, Robert Moorhead, M.B., Assistant-Physician at The Belfast Hospital for Sick Children; 9, Clifton Street, Belfast. (C. 1907–8.) (C.)

1901 Beddard, Arthur Philip, M.D., Senior Assistant-Physician and Pathologist at The West-London Hospital; 44, Seymour Street, W.

1902 Blaikie, James Brunton, M.B., Physician at The Hospital for Consumption, Margaret Street, W.; 80, Brook Street, W.

O.M. Bluck, Walter Cardy, M.B., Byford House, York Road, Southend-on-Sea.

1903 Braithwaite, John, M.D., Physician at The Devonshire Hospital and Buxton Bath Charity, 2, Hardwick Mount, Buxton.

1906 Brewerton, Elmore Wright, Ophthalmic Surgeon at The Metropolitan Hospital; Assistant Surgeon at The Royal Westminster Ophthalmic Hospital; 84, Wimpole Street, W.

Elected

O.M. Brightman, Frank, Aspley House, Broadstairs, Kent.

1901 Brown, John Henry, M.D., 14, Burngreave Road, Sheffield.

O.M. Browne, Hablot J. M., M.D., 7, Market Street, Hoylake, Cheshire.

1908 Bunch, J. L., M.D., Physician in Charge of the Skin Department at The Queen's Hospital for Children; 84, Park Street, W.

1904 Burgess, Harold, L., c/o Messrs. Worsfold and Hayward, Market Square, Dover, Kent.

1904 Burges, Richard, 4, Worcester Terrace, Clifton.

1901 *Burgess, William Milner, Ellerslie, 4th Avenue, Frinton-on-Sea, Essex. (C. 1904–5.)

O.M. Burghard, Fred. François, M.S., Surgeon at King's College Hospital and at The Paddington-Green Children's Hospital; 86, Harley Street, W.

1905 Burnet, James, M.D., Registrar at The Royal Hospital for Sick Children, Edinburgh; 6, Glengyle Terrace, Edinburgh.

1904 Burrows, Harold, M.B., 1, The Cams, Grove Road, Southsea.

1901 Burt, Albert Hamilton, 23, Palmeira Square, Brighton.

1907 Burton, Charles George, 91, Hertford Road, Lower Edmonton, N.

1902 Bythell, W. J. S., "Woodcliffe," Prestwich, Manchester.

1905 Campbell, Harry, M.D., Physician at The North-West London Hospital, and at The West End Hospital for Nervous Diseases; 23, Wimpole Street, W.

1902 Campbell, Henry Johnstone, M.D., Senior Physician at The Bradford Royal Infirmary. (C. 1907–8.)

1901 Campbell, Robert, M.B., Senior Surgeon at The Belfast Hospital for Sick Children; 21, Great Victoria Street, Belfast. (C. 1904–5, 1905–6.)

O.M. Carmichael, James, M.D., Consulting-Physician at The Royal Edinburgh Hospital for Sick Children; 22, Northumberland Street, Edinburgh. (C. 1901–2.)

Elected

O.M. Carpenter, George, M.D., Membre Correspondant de la Société de Pédiatrie de Paris, Physician at The Queen's Hospital for Children; late Physician at The Evelina Hospital for Sick Children; Editor and Founder of 'The British Journal of Children's Diseases'; 12, Welbeck Street, W. (C. 1900–1.) (S. 1901–2, 1902–3, 1903–4.) (Ed. 'Reports' 1900–1, 1901–2, 1902–3, 1903–4, 1904–5, 1905–6, 1906–7, 1907–8.) (C.C. 1907–8.) (W.)

O.M. Carre-Smith, Herbert L., 74, Holland Park Avenue, Holland Park, W. (C. 1902–3, 1903–4, 1904–5.)

1900 Cathcart, George C., M.B., Senior Assistant-Surgeon at The London Throat Hospital, 35, Harley Street, W.

O.M. *Cautley, Edmund, M.D., Physician at The Belgrave Hospital for Children; Physician at The Metropolitan Hospital; 15, Upper Brook Street, W. (C. 1900–1, 1901–2, 1902–3.) (S. 1904–5, 1905–6.) (C. 1906–7, 1907–8.) (C.)

1901 Cave, Edward John, M.D., Physician at The Royal United Hospital, Bath; 20, Circus, Bath.

O.M. Chaffey, Wayland C., M.D., Physician at The Royal Alexandra Hospital for Children, Brighton; 18, Palmeira Square, Hove, Sussex. (C. 1900–1, 1901–2, 1902–3.)

1901 Chaldecott, John Henry, Anæsthetist at St. Mary's Hospital and at The Metropolitan Throat, Nose, and Ear Hospital; 60, Harley Street, W.

O.M. Chapman, Charles William, M.D., Physician at The Hospital for Diseases of the Heart, Soho Square; 21, Weymouth Street, W. (C. 1903–4, 1904–5, 1905–6.) (C.)

1901 Cheyne, Sir William Watson, Bart., C.M., Surgeon at King's College Hospital, and at The Paddington-Green Children's Hospital; 75, Harley Street, W. (C. 1901–2.) (W.)

1908 Chisholm, R. A., M.B., Assistant-Physician at the Queen's Hospital for Children; 16, Weymouth Street, W.

1904 Clogg, H. S., M.S., Assistant-Surgeon at Charing Cross Hospital, and Surgeon at The Evelina Hospital for Sick Children; 143, Harley Street, W. (C. 1907–8.) (C.)

Elected

1905 Clutterbuck, L. A., M.D., Physician at The Welbeck Street Dispensary; Oakwood, Wentworth Road, Golders Green, nr. Hendon, N.W.

1902 Cockburn, Robert P., 64, The Avenue, Ealing, W.

1902 Collier, H. Stansfield, Surgeon to Out-patients at St. Mary's Hospital, and Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 57a, Wimpole Street, W.

1901 Collier, Mark Purcell Mayo, M.S., Senior Assistant-Surgeon at The North-West London Hospital; Consulting-Surgeon at The National Hospital for Diseases of the Nervous System; 133, Harley Street, W.

1901 Cooper, Percy Robert, M.D., "Glenthorne," The Downs, Bowdon, Cheshire.

1901 Corner, Harry, M.D., Brook House, Southgate, N.

1905 Cowper, W. P., 30, Frognal Lane, Hampstead, N.W.

1901 Cox, Joshua J., M.D., Physician for Diseases of Children at The Clinical Hospital, Manchester; 38, Deansgate, Manchester.

1902 Cross, Ernest, W., "The Limes," Wallwood Park, Leytonstone.

1901 Crossley, Leonard, M.B., "Beechwood," Arnside, Carnforth, Lancashire.

1902 Crowley, Ralph H., M.D., Physician at The Bradford Royal Infirmary; 116, Manningham Lane, Bradford.

1905 Cuff, Archibald, Surgeon at The Royal Infirmary, and Assistant-Surgeon at The Children's Hospital, Sheffield; 285, Glossop Road, Sheffield.

1904 Curl, Sydney Walter, M.D., 58, Crouch Street, Colchester.

1902 Curtis, Henry J., B.S., Assistant Surgeon at The Metropolitan Hospital, Kingsland Road; 58, Harley Street, W.

1901 Dawnay, Archibald Hugh Payan, Anæsthetist at The Royal Orthopædic Hospital; 126, Harley Street, W.

O.M. Deas, Frank, Thorndale, Merton Park, S.W. (C. 1905–6, 1906–7.)

Elected

O.M. Dent, Clinton T., M.C., Surgeon at St. George's Hospital and Consulting-Surgeon at The Belgrave Hospital for Children; 61, Brook Street, W. (C. 1902–3, 1903–4, 1904–5.)

1901 Dodd, Arthur Herbert, 4, Ventnor Villas, Hove, Sussex. (C.)

1901 Douglas, Arch. R., Assistant-Medical Officer and Deputy Superintendent at The Royal Albert Asylum, Lancaster.

O.M. Drew, Douglas, B.S., Surgeon at The Queen's Hospital for Children and at The Hospital for Women, Soho Square; 6, Wimpole Street, W. (C. 1907–8.)

1905 Dudgeon, Leonard S., Superintendent of Clinical Laboratory, Bacteriologist and Lecturer on General and Special Pathology in the Medical School at St. Thomas's Hospital; 6, Powis Garden, Bayswater, W.

1901 Dun, Robert Craig, M.B., Surgeon at The Liverpool Infirmary for Children; 6, Rodney Street, Liverpool.

1901 Dunlop, James, Aveley, Abington, Upminster, Essex.

1901 Dunn, W. J. Robertson, 46, Wellington Terrace, West Derby Road, Liverpool.

1906 East, C. H., M.D., St. Clare, Malvern.

1901 Edelsten, Ernest Alfred, M.B., 370, Brixton Road, S.W.

1904 Edmunds, Arthur, B.S., Assistant-Surgeon at The Paddington Green Children's Hospital; 30, Welbeck Street, W.

O.M. Edmunds, Walter, M.C., Surgeon at The Prince of Wales's Hospital, Tottenham; 2, Devonshire Place, W. (C. 1900–1, 1901–2, 1902–3.)

1905 Elliott, Andrew, M.D., Medical Registrar at The West London Hospital; 110, Gloucester Place, Portman Square, W.

1906 Elliott, Frank Percy, M.B., Medical Officer at The Walthamstow Public Dispensary, 113, Grove Road, Walthamstow.

1901 Ellis, W. McDonogh, M.D. 16, Eliot Vale, Blackheath, S.E.

1904 Emanuel, J. G., M.D., Physician to Out-patients at The Queen's Hospital, Birmingham; 47, Newhall Street, Birmingham. (P.S. 1907–8.) (C.)

Elected

1901 Evans, J. Howell, M.Ch., Assistant-Surgeon at The Cancer Hospital; 63, Grosvenor Street, W.

1901 Evans, Thomas Hy., 95, Shaw Street, Liverpool.

1904 Evelyn, William Arthur, M.D., Honorary Physician at The York County Hospital; 24, Micklegate, York.

O.M. Ewart, William, M.D., Senior Physician at St. George's Hospital and at The Belgrave Hospital for Children; 33, Curzon Street, Mayfair, W. (C. 1904–5, 1905–6, 1906–7.)

1901 Fisher, H. Wycliffe, "Rathescar," Greenbank Drive, Sefton Park, Liverpool.

O.M. Fisher, Theodore, M.D., late Pathologist at The Bristol Royal Infirmary, and late Physician to Out-patients at The Bristol Hospital for Sick Children; Assistant-Physician at The East London Hospital for Children; The Garples, Sidcup, Kent. (C. 1900–1.) (P.S. 1901–2, 1902–3, 1903–4.) (C. 1904–5.)

1906 Fitzwilliams, Duncan C. L., M.B., 64, Brook Street, W.

1906 Fordyce, A. Dingwall, M.B., Physician for Diseases of Children at The Newtown Dispensary; 19, Coates Crescent, Edinburgh.

1901 Forster, Henry John, 119, Victoria Road, Romford, Essex.

1903 Fortescue-Brickdale, John Matthew, M.D., Registrar and Pathologist at The Royal Hospital for Children and Women, Bristol; 52, Pembroke Road, Clifton, Bristol.

O.M. Fox, T. Colcott, M.B., Physician for Diseases of the Skin at Westminster Hospital, and at The Paddington-Green Hospital for Children; 14, Harley Street, W.

1905 Fox, W. S., 63, Grosvenor Street.

1907 Fraser, D. H., M.B., 166, Coldharbour Lane, S.E.

1901 *Frost, Edmund, M.D., "Chesterfield," Chesterfield Road, Eastbourne.

O.M. Fuller, Andrew, Local Government Board Inspector; "The Bushes," Chase Road, Oatlands Park, Weybridge, Surrey.

1906 Furber, Edward Price, Beechcroft, Oxted, Surrey.

1908 Gardner, Henry Willoughby, M.D., 23, St. John's Hill, Shrewsbury.

Elected

1901 George, Alfred Walter, M.D., Surrey House, Shoot-up Hill, N.W.

1904 Gerrish, Daniel S., Coningsby Villa, 332, Church Road, St. George, Bristol.

1900 Gervis, F. Harcourt, M.D., 1, Lyncroft Mansions, West Hampstead, N.W.

1908 Gibson, G. A., M.D., Physician at The Royal Infirmary, Edinburgh; 3, Drumsheugh Gardens, Edinburgh. (C.)

O.M. Giffard, Henry Edward, Denham House, Egham.

1907 Gilford, Sidney, M.B., 136, Oxford Road, Reading.

1905 Goldsmith, G. Harvey, M.D., Ophthalmic Surgeon at The Bedford County Hospital; Harpur Place, Bedford.

1901 Grant, J. Dundas, C.M., Surgeon at The Central London Throat and Ear Hospital; 18, Cavendish Square, W.

O.M. Gray, Alexander C. E., M.D., 7, Cromwell Place, South Kensington, S.W. (C. 1906–7, 1907–8.) (C.)

1901 Gullan, Archibald Gordon, M.D., 37, Rodney Street, Liverpool.

O.M. Guthrie, Leonard George, M.D., Physician at The Hospital for Epilepsy and Paralysis, Regent's Park; Senior Physician at The Paddington-Green Children's Hospital: 15, Upper Berkeley Street, W. (C. 1900–1, 1901–2, 1902–3.) (C. 1904–5, 1905–6, 1906–7.)

1907 Haig, T. B., M.D., 88, Dalston Lane, N.E.

1904 Harman, N. Bishop, M.B., Ophthalmic Surgeon at The Belgrave Hospital for Children; 108, Harley Street, W.

1907 Harvey, Alfred Wallace, B.A., 46, Adair House, Oakley, Chelsea, S.W.

O.M. Hawken, Giles Lockwood Lang, Surgeon at St. John's College, Hurstpierpoint, Sussex.

1901 Hawthorne, Charles Oliver, M.D., Physician at The Central London Ophthalmic Hospital: Assistant-Physician at The Royal Hospital for Children and Women; 63, Harley Street, W. (C. 1905–6, 1906–7, 1907–8.)

1904 Haynes, G. Secretan, 4, Trinity Street, Cambridge.

Elected

1906 Hett, G. Seccombe, M.B., 16, Palace Gardens Mansions, The Mall, Kensington, W.

1901 Heywood, Charles Christopher, M.B., Physician at The Manchester Children's Hospital and Assistant-Physician for Diseases of Children at The Manchester Northern Hospital; Winter's Buildings, St. Ann Street, Manchester.

1907. Higgs, Frederick W., M.B., 6, Hertford Street, Mayfair, W., Assistant-Physician at the Belgrave Hospital for Children.

1904 Hill, G. Wm., M.D., Surgeon for Diseases of the Ear at St. Mary's Hospital; 26, Weymouth Street, Portland Place, W.

1902 Hinds, Frank, M.D., Honorary-Surgeon at The Infirmary, Worthing; 17, Tennyson Road, Worthing. (C. 1904–5, 1905–6.)

1901 Hobhouse, Edmund, M.D., Assistant-Physician at The Sussex County Hospital; Physician at The Royal Alexandra Hospital for Children, Brighton; 12, Second Avenue, Hove. (C. 1903–4. 1905–6, 1906–7, 1907–8.)

1904 Hobson, H. Overton, M.D., 32, Upper Berkeley Street, W.

1905 Hollings, Guy B., M.D., 22, Endsleigh Gardens, N.W. (C. 1907–8.) (C.)

1907 Holmes, Thomas Edward, M.D., Portland Lodge, Knighton, Leicester.

1907 Holtzmann, W. W., M.B., London Hospital, E.

1901 Hopkinson, Albert, M.B., 2, Parsonage Road, Withington, Manchester.

1905 Howard, J. Russell, M.S., Assistant-Surgeon at The Royal Waterloo Hospital for Women and Children; 10, Weymouth Street, W.

1901 Hutchison, Robert, M.D., Assistant-Physician at The London Hospital, and at The Hospital for Sick Children, Great Ormond Street; 22, Queen Anne Street, W. (C. 1901–2, 1902–3. 1903–4.)

O.M. Hutton, David Mount, M.D., 5, Lulworth Road, Birkdale.

O.M. Hutton, Henry Richmond, M.B., Physician at The Manchester General Hospital for Children; 16, St. John Street, Manchester. (C. 1903-4, 1904–5, 1905–6.)

Elected

1904 imThurm, Robert McKenzie, Ailsa, Sydenham.

O.M. Jaffrey, Francis, Surgeon at St. George's Hospital; Surgeon at The Belgrave Hospital for Children; 33, Nottingham Place, W. (C. 1901–2, 1902–3, 1903–4.) (S. 1905–6, 1906–7.) (C. 1907–8.) (C.)

1901 James, Charles Alfred, 49, Stamford Hill, N.

1905 Jessop, Edward, 81, Fitzjohn's Avenue, Hampstead, N.W.

O.M. Jessop, Walter Hamilton Hylton, M.B., Ophthalmic-Surgeon at St. Bartholomew's Hospital; 73, Harley Street, W.

1901 Johnson, L. Capper, Lidgate Park Road, Roundhay, Leeds.

1902 Johnson, Raymond, Surgeon at University College Hospital and at The Victoria Hospital for Children; 11, Wimpole Street, W.

1904 Jones, A. Ernest, M.D., 13, Harley Street, W.

1903 Jones, W. Llewellyn, M.D., 58, Thomas Street, Merthyr Tydfil, South Wales.

1908 Jordan, Walter Ross, M.D., 91, Cornwall Street, Birmingham.

1905 Kellie, Kenneth H. A., M.B., 2, Chester Gate, Regent's Park, N.W.

1902 Kelson, William Hy., M.D., Assistant-Surgeon at The London Throat Hospital; 17, Cavendish Place, Cavendish Square, W.

O.M. Knapton, George, Leeswood House, Rhos, Ruabon, N. Wales.

1901 Lacy, Alexander Gairdner, "Saumarez," Sunninghill, Ascot, Berks.

O.M. Lambert, Sydney H. A., M.B., Gothic House, Harrow-on-the-Hill. (C. 1906–7.)

1901 Langdon-Down, Percival L., M.B., 47, Welbeck Street, W.

1900 Langdon-Down, Reginald Langdon, M.B., 47, Welbeck Street, W.

1905 Langmead, F., M.D., 74, Oxford Terrace, Hyde Park, W.

O.M. Lawson, Arnold, M.D., Assistant-Surgeon at The Royal London Ophthalmic Hospital, and Ophthalmic-Surgeon at The Children's Hospital, Paddington-Green; 12, Harley Street, W.

Elected

1905 Lee, H. E., M.B., Glen Aye, Chatswood, N.S.W.

O.M. Lee, Philip George, Surgeon at The Women and Children's Hospital, Cork; 25, St. Patrick's Hill, Cork. (C. 1906–7, 1907–8.) (C.)

1907 Leon, John F., M.D., Elmwood, Grove Road, Southsea, Hon. Assistant-Physician at The Royal Portsmouth and Gosport Hospital.

1901 Leslie, W. Murray, M.D., 74, Cadogan Place, Belgrave Square, S.W.

1904 Lett, Hugh, Ch.B., Assistant-Surgeon at The London Hospital and at The Belgrave Hospital; 48, Queen Anne Street, W. (C. 1906–7.) (S. 1907–8.) (S.)

O.M. Lewis, Percy G., M.D., Honorary Medical Officer at The Victoria Hospital, Folkestone; 22, Manor Road, Folkestone. (C. 1903–4, 1904–5, 1905–6.) (C.)

1904 Lingard, Alfred, M.B., late Imperial Bacteriologist to the Indian Government. (Travelling.)

1901 Livingstone, George R., 35, George Street, Dumfries, N.B.

1905 Low, V. Warren, B.S., Assistant-Surgeon at St. Mary's Hospital; 146, Harley Street, Cavendish Square, W.

O.M. *Lucas, R. Clement, B.S., Senior Surgeon and Lecturer on Surgery at Guy's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 50, Wimpole Street, W. (T. 1900–1, 1901–2, 1902–3, 1903–4, 1904–5, 1905–6, 1906–7, 1907–8.) (W. 1903–4.) (C.C. 1905–6, 1906–7.) (TT. 1907–8.) (C.)

1905 Maddox, R. H., M.B., Major I.M.S., Ranchi, Chota Nagpur, India; c/o Messrs. Thos. Cook & Sons, Ludgate Circus.

O.M. McCaw, John, M.D., Physician at The Belfast Hospital for Children; 74, Dublin Road, Belfast (C. 1901–2.)

1904 McGregor, John, M.D.

1905 McIlraith, C. H., M.D., 44, Half Moon Street, Herne Hill, S.E.

O.M. McKenzie, Dan, M.D., Holly Lodge, Leytonstone.

O.M. McKerron, Robert Gordon, M.D., Senior Physician at The Children's Hospital, Aberdeen; 1, Albyn Place, Aberdeen. (C. 1902–3, 1906–7, 1907–8.)

Elected

1901 McNab, Alexander Anderson, M.B., 15, Wesley Road, Armley, Leeds.

1900 Macalister, Charles John, M.D., Physician at The Liverpool Royal Southern Hospital; 35, Rodney Street, Liverpool. (C. 1901–2, 1902–3, 1903–4.)

1907 Mackenzie, John Baird, M.B., Ardlair, Spylaw Road, Edinburgh.

1901 Mackintosh, John Stewart, St. Ive, Platt's Lane, Hampstead, N.W.

1901 Mackintosh, William Alexander, M.B., 3, Park Terrace, Stirling. (C. 1904–5.)

1901 Maitland, Alfred Derwent, 1, Gloucester Villas, Hanwell, W.

1904 Makin, Frank Humphrey, M.B., c/o 'South African Register,' 80, Fleet Street, E.C.

O.M. Malcolm, John D., C.M., Surgeon at The Samaritan Free Hospital; 13, Portman Street, Portman Square, W.

1907 Mann, H. C. C., M.B., 8, St. Thomas's Street, S.E.

1907 Manuel, Alexander, M.B., 2, Marlborough Gate, W.

1908 Marshall, Charles Fred., M.D., 27, New Cavendish Street, W.

O.M. Marshall, Lewis W., M.D., Surgeon at The Children's Hospital, Nottingham; 42, The Ropewalk, Nottingham. (C. 1900–1, 1901–2, 1902–3.)

1908 Martin, Albert, M.D., late Examiner in Medicine and Surgery and Physiology at The Otago University, New Zealand, late Physician at The Wellington Hospital, late Surgeon at The Wellington Hospital, Surgeon-Major New Zealand Army Medical Corps; Wellington, New Zealand.

1907 Mathews, Paul, M.D., Resident Medical Officer, St Mary's Hospital, Manchester.

1900 May, W. Page, M.D., 9, Manchester Square, W.

1902 Maynard, J. Seymour, M.B., 4, Charles Street, St. James's Square, S.W.

1904 Midelton, William John, 72, Charminster Road, Bournemouth.

O.M. Milburn, Charles Henry, M.S., Surgeon at The Victoria Hospital for Children, Hull; 158, Anlaby Road, Hull. (C.)

Elected

1907 Miller, Reginald Henry, M.D., 22a, Cavendish Square, W.

1902 Milligan, Wyndham Anstruther, M.D., Registrar at The Hospital for Women, Soho Square; Pathologist at The Queen's Hospital for Children; 68, Park Street, Grosvenor Square, W.

1907 Milne, James A., Her Majesty's Hospital, Stepney, E.

1901 Milne, Robert, M.D., 32, Bow Road, E.

O.M. Moline, Paul F., M.B., Anæsthetist at The Victoria Hospital for Children, Chelsea; 42, Walton Street, Chelsea, S.W.

1901 Monsarrat, Keith Waldegrave, C.M., Surgeon at The Northern Hospital, Liverpool; 11, Rodney Street, Liverpool. (C. 1904–5.)

1901 Montgomery, W. P., B.S., Surgeon at The Children's Hospital, Manchester, Assistant-Surgeon at The Manchester Royal Infirmary; 15, St. John Street, Manchester.

1906 Moon, Edward Gibson, High Beach, Victoria Parade, Broadstairs.

1907 Moore, James Lennox Irwine, M.B., 30a, Wimpole Street, W.

1901 Morgan, Cecil Augustus, M.B., 6, Gloucester Place, Portman Square, W.

1902 Morgan, George, Senior Surgeon at The Children's Hospital, Brighton; 6, Pavilion Parade, Brighton.

1901 Morison, Alexander, M.D., Physician to Out-patients at The Great Northern Central Hospital, and at The Children's Hospital, Paddington-Green; 14, Upper Berkeley Street, W. (C. 1903–4, 1904–5, 1905–6.)

O.M. Mossop, Isaac, Surgeon at The Bradford Children's Hospital; Victor Lodge, Manningham, Bradford. (C. 1906–7.)

1905 Mounsey, R. E., Moorlands, Camberley, Surrey.

1902 Mummery, John Percy Lockhart, B.C., 10, Cavendish Place, W., Assistant-Surgeon at The Queen's Hospital for Children. (C. 1904–5, 1905–6, 1906–7.) (C.)

1901 Munro, Henry Acland, M.B., "Lulworth," Rushey Green, Catford, S.E.

1904 Murphy, J. Keogh, B.C., Surgeon to Out-patients at The Paddington-Green Children's Hospital; 9, Weymouth Street, Portland Place, W.

Elected

1904 Nash, Elwin H. T., Oulton House, Burnley Road, Accrington, Lancs.

1901 Nash, W. Gifford, Surgeon at The Bedford County Hospital; Clavering House, Bedford.

1905 Neave, Sheffield, Assistant-Physician at The Queen's Hospital for Children; Mill Green Park, Ingatestone, Essex.

1902 Newbolt, George P., Surgeon at The Royal Southern Hospital, Liverpool; 42, Catherine Street, Liverpool.

1908 Newsom, Morris Leonard, Surgeon at The Bristol Dispensary, Pathologist at The Hospital for Sick Children, Bristol; Avonlea, Queen's Park Road, Redfield, Bristol.

1901 Nisbet, J. Tawse, M.D., 7, Croxteth Road, Liverpool.

1901 Niven, Robert, Camberwell Infirmary, Brunswick Square, S.E.

1906 Nix, Edward J., M.D., Physician at All Saints' Children's Hospital; 11, Weymouth Street, W.

1906 Odgers, P. N. Blake, B.Ch., Assistant-Surgeon at The Northampton Royal Hospital; 16, Castilian Road, Northampton.

1901 Oldmeadow, Lloyd J. H., M.D., Kineton, Warwickshire.

1901 Oppenheimer, Heinrich, M.D., 63, Finsbury Pavement, E.C.

1904 Orme, Gilbert Edward, B.C., Lowfield, Clitheroe, Lancs.

1907 Parbury, W. K., Medical Officer of Health, Bedfordshire district, Thornbrook, Bedford.

1906 Parker, Arthur Percy, B.Ch., Surgeon at The Radcliffe Infirmary, Oxford; 2, Holywell Street, Oxford.

O.M. Parkinson, J. Porter, M.D., Physician at The London Temperance Hospital and Physician at The Queen's Hospital for Children; 57, Wimpole Street, W. (C. 1903–4, 1904–5, 1905–6.) (S. 1906 7, 1907–8.) (C.)

1902 Parkinson, Thomas W., M.D., 77, Sloane Street, S.W

1903 Parry, Robert H., Surgeon at The Victoria Infirmary and at The Royal Hospital for Sick Children, Glasgow; 3, Royal Crescent, Queen's Park, Glasgow. (C.) (1903–4.)

Elected

1907 Parsons, Leonard Gregory, M.B., 39, High Street, Bromsgrove, Worcestershire.

1901 Paterson, Alexander Gordon, M.D., South Lodge, Ascot, Berks.

1903 Pearson, S. Vere, M.B., Physician at Mundesley Sanatorium, Norfolk.

1901 Pendlebury, Herbert S., B.C., Surgeon at The Royal Hospital for Women and Children, Assistant-Surgeon at St. George's Hospital; 44, Brook Street, W.

O.M. *Penrose, Frank, M.D., late Physician at St. George's Hospital and at The Hospital for Children, Great Ormond Street; Wick House, Downton, Salisbury, Wilts.

1901 Pernet, George, M.D., Assistant at the Skin Department, University College Hospital; 94, Harley Street, W. (C. 1903–4, 1904–5, 1905–6.) (C.)

1907 Perram, Charles Herbert, M.D., Assistant-Physician at The County Hospital, Bedford; 55, Bromham Road, Bedford.

1901 Perry, Herbert, M.D., Assistant-Physician at The General Hospital, Birmingham, Physician to Out-patients at The Children's Hospital, Birmingham; 54, Newhall Street, Birmingham. (C. 1905–6, 1906–7, 1907–8.)

O.M. Peskett, A. W. Chalmers, M.B., 4, Clermont Road, Preston Park, Brighton.

1902 Phillips, Hugh, M.B., 28, Palace Court, W.

1905 Pike, Douglas R., M.B., Stalmine Lodge, Stalmine, Poulton-le-Fylde, Lancashire.

1904 Piper, Francis Perris, M.B., King's Leigh, Tankerton, Whitstable.

1902 Potter, Bernard Elwell, M.B., Resident Medical Officer at St. George's Dispensary, Hanover Square; 37, Park Street, W.

1901 Potts, William A., M.D., 160, Hagley Road, Birmingham.

1904 Powell, Hugh Falkenberg, 1, Seagrove Place, Pittville, Cheltenham.

1904 Poynton, Frederick J., M.D., Assistant-Physician at University College Hospital, and at The Hospital for Sick Children, Great Ormond Street; 1, Harley Place, Harley Street, W. (C. 1906–7, 1907–8.)

1901 Pratt, Eldon, M.D., Henfield, Sussex.

Elected

1901 PRITCHARD, GEORGE ERIC CAMPBELL, M.D., 63, Harley Street, W.

1901 QUENNELL, ARTHUR, Brentwood, Essex.

1905 RANDALL, MARTIN, M.D., The Lodge, Leopold Road, Wimbledon, S.W.

1901 RAW, NATHAN, M.D., Mill Road, Liverpool.

1901 RAY, JOHN HOWSON, Ch.M., Assistant-Surgeon at The Salford Royal Hospital; Surgeon at The Children's Hospital, Pendlebury; 11, St. John Street, Manchester.

1901 RENDEL, ARTHUR BOWEN, M.B.

1901 RHODES, JOHN MILSON, M.D., Didsbury.

1903 RIDLEY, J. BROOKE, M.D., 53, Dingwall Road, Croydon.

1908 RISCHBIETH, H., B.C., London Hospital, E.

1906 ROBERTS, CHARLES, M.B., Surgeon at The Manchester Children's Hospital; 15, St. John Street, Manchester.

O.M ROBINSON, HENRY BETHAM, M.S., Assistant-Surgeon at St. Thomas's Hospital and Surgeon at The East-London Hospital for Children; 1, Upper Wimpole Street, W. (C. 1901–2, 1902–3, 1903–4.)

O.M. ROGERS, BERTRAM, M.H., M.D., Physician at The Hospital for Sick Children and Women, Bristol; 1, Victoria Square, Clifton, Bristol. (C. 1907–8.) (C.)

1901 ROSE, SAMUEL FRANK, Assistant-Surgeon at The National Dental Hospital; Dental-Surgeon at The Queen's Hospital for Children; 25, Weymouth Street, Portland Place, W.

O.M. ROTH, BERNARD, Orthopædic-Surgeon at The Royal Alexandra Hospital for Children, Brighton; 38, Harley Street, W.

1902 *ROXBURGH, ALEX. BRUCE, B.Ch., Surgeon at The Poplar Hospital, and Assistant-Surgeon at The London Hospital; 7, Henrietta Street, Cavendish Square, W.

1901 RUMBOLL, SYDNEY, Cavendish House, Leeds.

1904 SALE-BARKER, HORACE JOHN MAURICE DRUMMOND, Medical Officer in Charge X-ray and Electrical Departments at Westminster Hospital; 10, Bentinck Street, W.

Elected

1904 Sawyer, James E., M.D., Casualty Assistant Physician at The General Hospital, Birmingham; Physician to Out-patients at The Children's Hospital, Birmingham; 93, Cornwall Street, Birmingham. (P.S. 1907–8.) (C.)

1908 Sawyer, Sir James, M.D., Consulting Physician at The General Hospital, Birmingham, 31, Temple Row, Birmingham.

1904 Scofield, H. C. Lingham, M.B., "Northwood," Dulwich Grove, North Dulwich, S.E.

1904 Scott, W. J., M.B., Clifton House, Sheet Street, Windsor.

O.M. Shuttleworth, George E., M.D., late Medical Superintendent Royal Asylum for Imbeciles, Lancaster; Medical Examiner defective children, Willesden School Board; Medical Expert (Metropolitan Asylums Board) Rochester House Inst., Ealing; 27, New Cavendish Street, W. (C. 1900–1, 1901–2, 1902–3.)

1901 Simson, Henry J. Forbes, 36, Grosvenor Street, W.

1901 Skelding, Henry, B.C., Surgeon at The Bedford County Hospital; St. Loyes, Bedford. (C. 1904–5, 1905–6.) (P.S. 1906–7.) (C. 1907–8.) (C.)

1907 Smallwood, E., The Bays, Knight's Hill, West Norwood.

1901 *Smith, Frederick John, M.D., Physician at The London Hospital; 138, Harley Street, W.

1908 Smith, P. Maynard, 1, Spanish Place, W.

1901 *Smith, Travers, R.M., M.B., 13, Kilburn Park Road, N.W.

1904 Smith, William R., M.D., Professor of Forensic Medicine at King's College, London; 74, Great Russell Street, Bloomsbury Square, W.C.

1901 Smyth, Sidney B., M.B., Physician at The Hospital for Sick Children, Belfast; Ulsterville House, Lisburn Road, Belfast. (C. 1903–4.)

O.M. Spokes, P. Sidney, Dental-Surgeon at University College Hospital; 4, Portland Place, W.

1903 Spriggs, Edmund Ivens, M.D., Assistant-Physician and Lecturer on Pathological Chemistry at St. George's Hospital, Assistant-Physician at The Victoria Hospital for Children and at The City of London Chest Hospital; 48, Bryanston Street, Portman Square, W. (S.)

1904 Spurway, John, M.B., 79, Brondesbury Road, Kilburn, N.W.

Elected

1901 Staveley, William H. C., 24, Sloane Gardens, S.W.

O.M. Stephenson, Sydney, C.M., Ophthalmic-Surgeon at The Evelina and The Queen's Hospitals for Children; Editor and Founder of 'The Ophthalmoscope'; 33, Welbeck Street, W. (S. 1900–1, 1901–2, 1902–3.) (C. C. 1903–4, 1904–5.) (C. 1905–6.) (C.)

O.M. Stewart, Hugh M., M.D., "Dyffryn," 123, Thurlow Park Road, Dulwich, S.E.

O.M. Stiles, Harold J., C.M., Surgeon at The Royal Edinburgh Hospital for Sick Children; 8, St. Colme Street, Edinburgh. (C. 1900–1, 1902–3, 1903–4.) (P.S.)

O.M. Sunderland, Septimus P., M.D., Physician at The Royal Hospital for Children and Women; Obstetric-Physician at The French Hospital; 11, Cavendish Place, W.

O.M. Sutherland, George A., M.D., Physician at The Paddington-Green Children's Hospital; Assistant-Physician at The North-West-London Hospital; 29b, Wimpole Street, W. (C. 1900–1, 1901–2, 1902–3.) (S. 1903–4, 1904–5.) (C. 1905–6, 1906–7, 1907–8.)

1904 Swainson, James Midleton Graham, Assistant-Surgeon at The Queen's Hospital for Children.

1907 Tallent, J. H., M.B., Walton Lodge, Chislehurst, Kent.

1901 *Targett, James H., M.S., Assistant-Obstetric-Surgeon at Guy's Hospital; 19, Upper Wimpole Street, W.

1901 *Taylor, Frederick, M.D., Consulting-Physician at Guy's Hospital; Consulting-Physician at The Evelina Hospital for Sick Children; 20, Wimpole Street, W. (C. 1901–2, 1903–4.) (TT. 1907–8.) (W. 1906–7.)

O.M. Taylor, James, M.D., Physician at The National Hospital for Paralysed and Epileptic, Queen Square, and Physician at The Queen's Hospital for Children; 49, Welbeck Street, W. (C. 1901–2, 1902–3, 1903–4.)

1906 Telford, E. D., Surgeon at The Children's Hospital, Manchester; 8, St. John Street, Manchester.

1905 Telling, W. H. Maxwell, M.D., Assistant-Physician at The General Infirmary and at The Hospital for Women and Children, and Physician at The Dispensary, Leeds; 29, Park Square, Leeds. (C. 1907–8.) (C.)

1908 Thompson, Harold Theodore, M.D., Assistant-Physician at The London Hospital and at The Hospital for Sick Children, Great Ormond Street, and Physician at The Poplar Hospital, 35, Harley Street, W.

Elected

1901 Thorne, W. Bezly, M.D., 2, Harley Street, W.

1907 Thorpe, G. E. Knight, 6, Tafton House Road, Sheffield.

1908 Torrens, James Aubrey, St. George's Hospital.

1901 Trout, J. H. Ensor, 412, Monument Road, Birmingham.

O.M. Tubby, Alfred H., M.S., Surgeon at Westminster Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; Surgeon at The National Orthopædic Hospital; 68, Harley Street, W. (C. 1900–1, 1901–2.) (C.C. 1902–3.) (C. 1904–5, 1905–6, 1906–7.) (C.)

1903 Tullis, George Augustus, M.D., Denstone, Christ Church Road, Winchester.

1901 Turnbull, George Lindsay, M.D., 76, Ladbroke Square, W. (C. 1906–7, 1907–8.) (C.)

1901 Turner, H. Stanley, "Balgownie," 211, Bridge Road, Battersea, S.W.

1904 Turner, Philip, M.S., Assistant-Surgeon at The Belgrave Hospital for Children; 8, St. Thomas's Street, S.E.

1902 Walker, J. W. Thomson, C.M., Assistant-Surgeon at The North-West London Hospital, Assistant-Surgeon at St. Peter's Hospital for Stone; 30, Queen Anne Street, W. (C. 1903–4, 1904–5, 1905–6.)

1901 Wallace, Joseph B., M.B., 117, North Side, Clapham Common, S.W.

1905 Wareham, Sidney James, Surgeon at St. Mary's Children's Hospital, Plaistow; Raymond House, Plashet Road, Upton Park, E.

1907 Warren, Richard, M.Ch., Surgical Registrar at The London Hospital; Assistant-Surgeon at The East-London Hospital for Children, Shadwell, E; London Hospital, E.

1901 *Weber, F. Parkes, M.D., Physician at The German Hospital, Dalston; Assistant-Physician at The North-London Hospital for Consumption; 19, Harley Street, W. (C. 1905–6.)

1908 Wells, J. E. B., Haddesdon, Herts.

1902 Westmacott, Frederick H., Aural-Surgeon at The Manchester Children's Hospital, Pendlebury; 8, St. John Street, Manchester.

Elected

1901 Whipham, Thomas Rowland C., M.D., Physician at The Evelina Hospital for Sick Children; Assistant-Physician at The Prince of Wales' General Hospital, Tottenham; 37, Park Street, W. (C. 1907–8.) (C.)

1906 White, Sinclair, M.D., Ranmore, Sheffield.

1901 Whitelocke, Richard H. A., C.M., Surgeon at The Radcliffe Infirmary, Oxford; 6, Banbury Road, Oxford. (C. 1901–2, 1902–3, 1903–4.) (P.S. 1904–5, 1905–6.) (C. 1906–7, 1907–8.) (C.)

O.M. Wilks, Joseph Henry, B.C., Surgeon at The Children's Hospital, Sheffield; "Fairfield," Sharrow, Sheffield.

O.M. *Williams, Dawson, M.D., Physician at The East-London Hospital for Children, Editor of 'The British Medical Journal'; 6, Catherine Street, Strand, W.C. (C. 1900–1, 1901–2.)

O.M. Williams, Edward Cecil, M.B., Physician to Out-patients at The Bristol Royal Hospital for Sick Children; 159, Whiteladies Road, Clifton, Bristol.

O.M. Willey, Charles H., C.M., Surgeon at The Sheffield Children's Hospital; Wostenholm Road, Sharrow, Sheffield. (P.S. 1900–1.) (C. 1901–2.)

1904 Wilson, Geoffrey R., Ongar, Essex.

O.M. Wilson, Kemlo R. M., M.D., Hanwell, W.

1902 Wood, Edward, Stamford Lodge, Waverley Road, Enfield, Middlesex.

1901 Woods, John Francis, M.D., Medical-Superintendent at The Hoxton House Asylum, N.; 29, Queen Anne Street, W.

1902 Wright, George A., M.B., Surgeon at The Manchester Royal Infirmary; Surgeon at The Children's Hospital, Pendlebury, Manchester; 16, St. John Street, Manchester.

1908 Yearsley, Percival Macleod, Senior Surgeon at the Royal Ear Hospital; Education Department, L.C.C.; 31, Harley Street, W.

⁂ *It is requested that any inaccuracy or inadequate description in the foregoing list may be notified to the Secretary of The Royal Society of Medicine, 20, Hanover Square, W. Members will greatly facilitate the work of the officials by examining the entries occurring opposite their names.*

REPORTS

OF

THE SOCIETY

FOR THE

STUDY OF DISEASE IN CHILDREN.

SESSION of 1907-8.

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1. ASSOCIATED MOVEMENTS OF UPPER EYELID AND JAW.

By SYDNEY STEPHENSON, C.M.

A FAIR number of cases have now been reported of synkinesis between the jaw and of an upper lid affected with ptosis. The peculiarity of the following case was that the abnormal association involved not the lid with ptosis, but the unaffected eyelid. The facts are as follows :

Charles G—, aged 9 years. Brought to the Evelina Hospital on September the 10th, 1907, on account of an attack of acute infective conjunctivitis, due to the Koch-Weeks' bacillus. According to the mother's statement, the child, when 9 months old and still in arms, was dropped on a step, with the consequence that his skull was dented in. His left eye has always been "smaller" than his right one, and since the accident mentioned his right upper lid has twitched when the child eats.

Present state.—Slight ptosis on left side. When the lad eats or chews the right upper lid jerks rhythmically up and down, the left upper lid showing no such associated movements. The same thing occurs when the mouth is opened or shut, or when the lower jaw is moved from side to side. No movements of the lid occur when the eyes are turned in various directions. Neither is there any pupillary anomaly. The patient has a peculiar physiognomy, and his skull exhibits a transverse depression behind the frontal bone.

Remarks.—My case falls into line with those of Fuchs, Fraenkel, and Eales, where, although there was no ptosis, a synkinetic association existed between one upper eyelid and the lower jaw. An abstract of these particular cases, together with a discussion of abnormal associated movements of the eyelids in general, will be found in a communication by Walter W. Sinclair, of Ipswich, in the 'Ophthalmic Review' for 1895, p. 307.

These "jaw-winking" movements, as they have been called, have been accounted for in several ways. The currently received explanation assumes that the levator palpebrarum, ordinarily supplied by the third nerve ucleus, receives in these cases an additional supply from the fifth nerve nucleus, which also innervates the muscles of mastication. This was the explanation put forward by a committee of the Ophthalmological Society appointed to examine into the first case of the kind reported in England, namely, one exhibited by R. Marcus Gunn in 1883 ('Transactions of the Ophthalmological Society,' vol. iii, 1883, p. 286). N. B. Harman, however, adopts another view (*ibid.*, vol. xxiii, 1903, p. 369). He regards the "jaw-winking" movement as an atavistic anomaly, in which there is an association of the jaw muscles with the migrated spiracle muscles.

(*Exhibited October the* 18*th*, 1907.)

Discussion.

The Chairman (**Mr. R. Clement Lucas**) asked what was the cause of the ptosis and of the third nerve trouble with which the condition was associated. The case was a very interesting one and extremely rare. One knew that there were certain associated movements which were not always necessarily the direct results of the same nerve supply. Movements sometimes took place in associated ways as the result of habit.

Dr. L. A. Clutterbuck asked whether the condition might not be possibly due to associated stimulation of the sympathetic. The upper eyelid was partially supplied from the sympathetic, and congenital ptosis was often due to affection of that rather than of the third nerve.

Mr. Sydney Stephenson, in reply, said that the ptosis was congenital. Certain post-mortem examinations of cases of congenital ptosis had shown that the levator palpebræ superioris was absent, or ill-developed, or malplaced.

2. TUBERCULOSIS OF THE IRIS AND CILIARY BODY, SHOWN WITH REFERENCE TO CALMETTE'S OPHTHALMO-REACTION.

By SYDNEY STEPHENSON, C.M.

May B—, aged 12 years, admitted into the Queen's Hospital for Children on August the 2nd, 1907, suffering from an affection of the left eye of about six months' duration.

Personal history.—Patient has had the usual childish ailments. Her left eye was noticed by the mother to be inflamed in the early part of February last; the sight became bad and the eyeball was noticed to be cloudy.

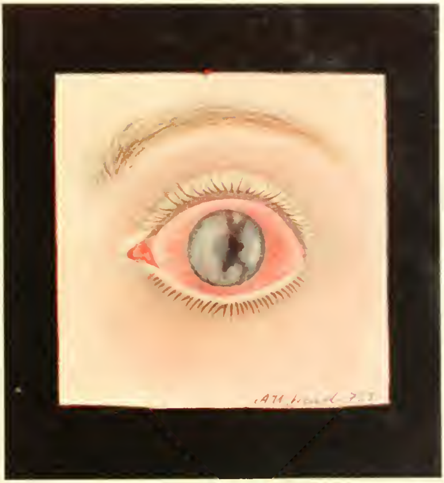
Family history.—The other two children in the family are stated to be healthy. A paternal aunt died from phthisis pulmonalis. The mother has had no miscarriages or children born dead.

On examination.—Anterior chamber of the left eye more or less filled with solid-looking, yellowish-grey exudation, so that a faint indication of the position of the pupil only could be obtained. The eye was reddened, flushed easily, and dreaded light somewhat. Tension was slightly minus; sight was equal only to counting the outspread fingers when held close to the patient's face. The child was well-nourished (3 st. 9 lbs.), and presented no signs either of syphilis or tubercle, with the possible exception of one enlarged gland at the angle of the jaw on the left side. An X-ray photograph of the chest (exhibited) revealed no morbid changes in lungs or mediastinum. Temperature 98·6° F. The appearance of the eye, however, was so characteristic that despite the slender evidence of tubercle I did not hesitate to diagnose tuberculosis of the iris and ciliary body, of the form described in the text-books as "conglomerate" or "massed" tubercle.

Progress.—The tuberculous nature of the ocular process was demonstrated by the general reaction that on two occasions followed the injection of $\frac{1}{1000}$ mgrm. of Koch's tuberculin T.R. It was further attested by the adoption of Calmette's test. On September the 22nd a drop of Calmette's serum instilled into the sound eye produced in nine hours a well-marked local reaction, which persisted for twenty hours.

In order to complete the case it should be added that in spite of repeated physical examination no other sign of tubercle, beyond that already mentioned, has yet been found. Although the temperature oscillates about the normal line, yet there is usually a slight rise towards night, 99·8° F. being the highest recorded in a series of observations extending from admission to the present time. The pulse-rate, usually about 90, has reached upwards of 100 on eighteen occasions only, although taken night and morning since admission. Respirations range from 16 to 24 a minute, and once alone have exceeded the last-named figure. Successive weights have been: 3 st. 9 lbs. (on admission), 3 st. 9½ lbs., 3 st. 10½ lbs., 3 st. 11¼ lbs., 3 st. 11 lbs., 3 st. 12 lbs., 3 st. 13 lbs., and 4 st. (September the 25th, 1907).

PLATE VII.



Mr. Sydney Stephenson's Case of Tuberculosis of the Iris and Ciliary Body

Adlard & Son, Impr.

Remarks.—The present case, although interesting and uncommon in itself, is shown more especially because it is one where Calmette's so-called ophthalmo-reaction was obtained. What is the ophthalmo-reaction? Some four months ago Professor Calmette, Director of the Pasteur Institute at Lille, announced that he had found a simple way of diagnosing tubercle in any part of the body ('La Presse Medicale,' June the 19th, 1907) by dropping into one eye a 1 per cent. watery solution of dried tuberculin. Neither ordinary tuberculin nor Koch's *Alttuberculin* must be employed, inasmuch as their contained glycerine is apt to produce irritation of the eye and thereby to mask any proper reaction. Now in healthy subjects the result of the solution of tuberculin is *nil.* The tuberculous, however, show a local reaction, now known as the ophthalmo-reaction of Calmette. Thus, three to five hours after the tuberculin has been used, the eye becomes congested, and before long presents the signs of an acute infective conjunctivitis, which may be mild, medium, or severe as regard its clinical characters. The inflammation may last for a few hours only, or, as in cases I have seen, persist for several days. The plan appears to be free from danger, local or general. It is delightfully simple, since its employment does not imply confinement to hospital. Lastly, it is important to note that Calmette's results have been confirmed by many observers, including Prouff, Grasset and Rimbaud, Letulle, Comby, Dufour and Buslé, Grasset, Montagnon, Bazy, Métraux, and Ferrand and Lemaire. My own experiences with the Calmette ophthalmo-reaction will be found by those interested in the subject in the current issue of the "British Medical Journal," October the 19th, 1907.

(*Exhibited October the 18th,* 1907.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said the pipette exhibited by Mr. Stephenson was most excellently prepared, and the Society was greatly indebted to that gentleman for bringing forward such a very modern form of test, one which was only a few months old, to demonstrate the presence of tuberculosis in the body generally. It showed the advantage of such a Society, in which all departments of the profession were represented, and by which members were able to learn the latest discoveries in relation to many diseases.

Dr. Edmund Cautley asked whether the substance produced any reaction whatever in normal subjects. He gathered from Mr. Stephenson that it did not, but he, Dr. Cautley, believed that Calmette in his original paper stated that a slight reaction occurred in normal people, of course not so severe as in tuberculous patients.

Mr. Sidney Stephenson, in reply, said that in his cases there was no reaction in people in whom there was no reason to suspect tuberculosis. Still, one or two people who had written on the subject had referred to a slight reaction in normal subjects. He had not seen it, and he thought the reaction in such must be very insignificant. Explanations of the action of the tuberculin had been offered, but he did not propose on the present occasion to go into them. But the principle involved in the application of Calmette's serum had been adopted in the diagnosis of typhoid fever.

3. A CASE OF MULTIPLE LUPUS VULGARIS.

By GEORGE PERNET, M.D.

This case, a girl aged 6 years and 9 months, had been previously brought before the Society by Dr. Poynton* on account of an acne scrofulosorum. That was in March of last year. About five months after that the child had an attack of measles, which according to the mother had cleared off the acne scrofulosorum. But three months later patches of Lupus vulgaris appeared on the left upper arm, the outer side of the right thigh and about the left ala nasi. Lupus vulgaris multiplex following measles was not uncommon, as many as fifty foci of lupus having been observed in this connection. The interest of the present case was the sequence of events : (1) Acne scrofulosorum developing in a child with large cervical glands and a marked history of phthisis on the mother's side; (2) measles; (3) multiple lupus, thus confirming clinical observations as to the tuberculous nature of acne scrofulosorum. (*Exhibited October the* 18*th*, 1907.)

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* See 'Reports' for 1906, vol. vi, p. 209.

4. A CASE OF LUPUS VULGARIS AS A RESULT OF DIRECT INOCULATION.

By GEORGE PERNET, M.D.

A girl, aged nine years, who, six months before being first seen (September the 20th, 1907), had been scratched with the finger-nail by another little girl in play. The scratch, which was on the face just below the right orbit, had not healed after three months and had then increased in size. The lesion was now ½ inch in length and about ⅛th inch wide, slightly scaly, and showing distinct nodules with glass-pressure. The appearances were now slightly altered owing to a few Finsen-light exposures.

(*Exhibited October the* 18*th*, 1907.)

Discussion.

The Chairman (Mr. R. C. Lucas), said that many years ago, when he was much interested in lupus and its treatment, he wrote an article on its treatment by scraping, which brought him so many cases, chiefly from the various workhouses in the country, even from as far as Anglesey, that he omitted mention of that article in the 'Medical Directory.' Among the many cases of lupus he saw, there were several which were the direct result of inoculation. One of them was a policeman, who stood at the corner of St. Thomas' Street in the Borough. One night he was taking a man into custody, when he struck him on the mouth and injured his own knuckle. From that little tooth mark on the knuckle lupus spread all over the back of the hand, and it was eventually cured by scraping. Another man was bitten on his forefinger, and from that lupus extended down the forefinger on to the back of the hand. That was cured in a like manner. Another patient, a child, was circumcised, and following the operation by a Rabbi lupus spread over the surface of the penis and pubes. That was also scraped. It was impossible to say what was the condition of the persons by whom the wounds were made, and he asked Dr. Pernet whether in his case he had been able to trace the source of inoculation, and whether the child who scratched the other was tuberculous, or lived in a house in which the disease existed. One way in which the condition was spread in the same person was by scratching. He had seen many cases of multiple lupus in children, and there was strong evidence that in many of the cases the disease was within the scratching area. After an exanthem such as measles, during the desquamative period there would be a great tendency to scratch.

Dr. Pernet, in reply, said he wanted to see the child who inoculated the present patient, but she had not come. His object had been to go into that point. He would endeavour to find out some facts about the people in the house. The present patient was not phthisical. He had seen several inoculation cases of lupus in adults, but those had generally been instances of verrucose lupus. Not long ago he had seen a case of a phthisical man who had scratched his knuckle, and in order to clean it used his own saliva. That started a regular lupus verrucosus on the back of the hand, which was got rid of by scraping and X rays. Another case was that of a woman who had been attending her phthisical husband; she had a similar verrucose lupus extending over the back of her hand and up the arm, and later she developed pulmonary tuberculosis, which she had also contracted from her husband. A medical student, while working in the laboratory with a tubercle bacillus culture, scratched his head, and the scratch was followed by lupus vulgaris of the part scratched. It was clearly inoculated by the culture.

5. A CURIOUS CASE OF INFANTILE PARALYSIS.

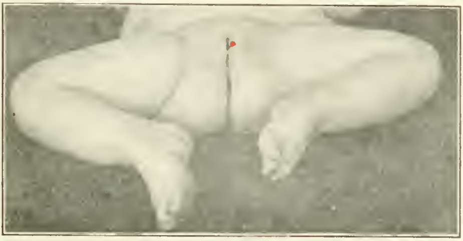
By P. LOCKHART MUMMERY, B.C.

Child aged 18 months. Quite healthy till last November, when it was very ill. During the illness it was noticed that the child lost the use of all its limbs, though the arms were only partly affected.

Since then it has recovered the use of its arms, but the flexor action of the fingers of the right hand is not quite normal.

There is very slight extensor power in the right thigh but none in the left limb. The interesting point about the case is that all the adductors on both sides are completely paralysed, a very unusual condition in infantile paralysis (Fig. 1). As a result, when the child is placed on her back the thighs fall outwards flat on the bed in a position of extreme abduction.

Fig. 1.



There is a considerable degree of secondary shortening of the non-paralysed muscles. Suggestions as to treatment of the deformity are invited.

(*Exhibited October the 18th,* 1907.)

6. A CASE OF GENU RECURVATUM—CONGENITAL.

By P. LOCKHART MUMMERY, B.C.

Child aged 3 months. Quite healthy except for the condition of the legs. It was born with the knees bending the wrong way.

Both legs are bent forward in an over-extended position to an angle of about 30 degrees.

It is impossible to flex the knees beyond the straight line. The patella cannot be felt on either side.

(*Exhibited October the 18th,* 1907.)

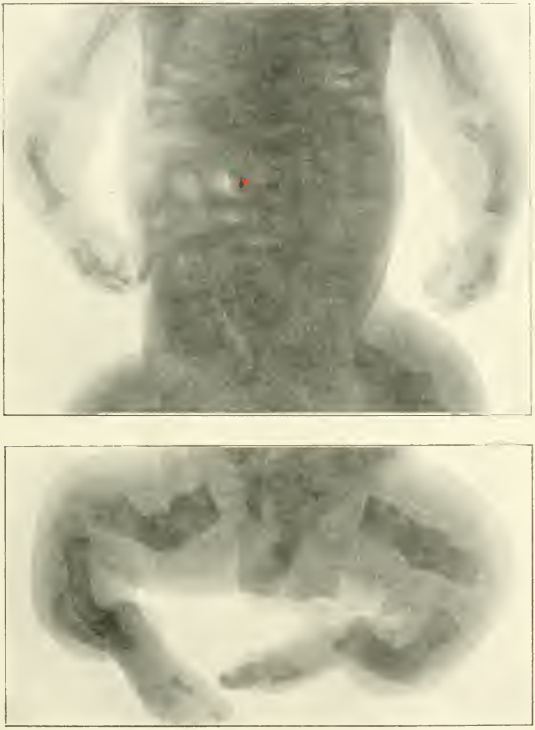
7. A CASE OF OSTEOGENESIS IMPERFECTA.

By F. LANGMEAD, M.D.

J. E—, a male infant, now aged 33 days, was brought to the Paddington Green Children's Hospital when he was four days old, because the limbs had been noticed to be bent at birth and were painful when moved. His birth was eventful. The mother believing herself to be about seven and a half months pregnant, and unaware of the onset of labour, was bathing other children in the kitchen when she felt a severe labour pain, and almost directly the head at the vulvar orifice. She held it back with her hand and got quickly to bed, where immediately the child and after-birth were expelled. It was soon noticed that the child's legs and arms were crumpled up, and for this it was brought to the hospital.

Family history.—The father and mother are quite well. There are nine other children also well. No miscarriages. No history pointing to syphilis. When examined at the hospital it was at once evident that he was strikingly and symmetrically deformed. The legs are sharply bent, almost to a right angle, a little below the middle of the bones, and the thighs, a little above the middles of the femora. Both forearms are bent about midway from elbows to wrists. The humeri are also slightly curved. In some places definite amniotic dimples can be seen. There are two in front of the bend in the left leg, one on the external and one on the internal aspect of the limb, and two more in the right lower extremity, one on the outer aspect of the bend in the leg and one on the posterior aspect of the junction of the upper and middle thirds of the thigh. The post-anal dimple is well defined. The skull is imperfectly ossified, especially about the lambdoid suture on the right side, where the bone is little more than membrane. A skiagram taken on the day of admission showed conclusively that the bending of the leg bones was due to fractures, and other skiagrams subsequently showed that this was true also of the arms, excepting the left radius and ulna, which are merely

Fig. 2.



curved. There are fractures of both tibiæ and fibulæ, of each femur in two places, of the right humerus and of the right radius and ulna, eleven in all, and bending of the left radius and ulna. About some of the fractures there is very marked callus formation, especially about those of the femora and leg bones. The fracture of the right humerus is evidently recent. The shadow given by the bones is no less dense than normal. The patient is a healthy-looking baby, and apart from the multiple fractures is apparently in normal health. His hair, however, is somewhat wiglike.

There can be no doubt that the fractures were intra-uterine, since the skiagram taken a few days after birth shows a considerable amount of callus, and the presence of amniotic dimples affords additional conclusive proof.

(*Exhibited October the* 18*th*, 1907.)

Discussion.

Mr. Hugh Lett said he was very much interested in the case, because it almost exactly resembled one which he showed before the Society last session. The history was almost exactly the same as in the present case. There was a history that the legs were noticed to be bent soon after birth; the labour was easy, and there was a complete absence of anything in the history which would lead to the supposition that it was hereditary. There was also imperfect ossification of the skull. In his case all the long bones, with the exception of the ribs and clavicles, were fractured and bent to almost as complete an angle as Dr. Langmead had described. At first it was thought to be rickets only, but when skiagrams were taken they showed that the lesions were the result of fractures, from the amount of callus thrown out. In his case there was no evidence of marked atrophy of bones, and their density seemed to be about the same as in a normal child of that age. In about 15 per cent. of the cases there was a history of other children in the same family being affected, or some of the relatives. He asked whether in the present case there was any evidence of hydrocephalus. There was in his case, and he believed it was a fairly frequent accompaniment. In his own case there was no evidence or history of syphilis. He did not know whether the case was at present living.

Dr. F. Langmead, in reply, said there was no sign of hydrocephalus, nor was there anything in the family history to suggest that it was hereditary.

8. A CASE OF CONGENITAL PTOSIS TREATED BY MOTAIS' OPERATION OF TENDON TRANSPLANTATION, VERY SLIGHTLY MODIFIED.

By SYDNEY STEPHENSON, C.M.

In general, the number of operations devised for the relieve of a given condition is in inverse ratio to their efficacy. To no condition of the eye does this truism apply with more force than to ptosis. The text-books abound with descriptions of surgical procedures which depend upon several different principles. These range, on the one hand, from the simple excision of skin, with or without a piece of the orbicularis muscle, as in v. Graefe's operation, to complicated plastic operations, such as that devised by Panas, on the other. Between these two extremes lie a host of other operations, such as the buried sutures of Pagenstecher, Dransart, Mules, Hess, Worth, Harman, and others; exsection or shortening of the levator palpebræ superioris, as carried out by Snellen, H. Wolff, and de Lapersonne respectively. Practically all these procedures have yielded good results in the hands of the surgeon who devised them, and some in the hands of other people.

Speaking for myself, I felt no confidence in obtaining a good result until I adopted the beautiful operation devised by Motais, of Angiers, which was first described at the French Academy of Medicine on March the 24th, 1903.* Briefly, Motais proposed to supplement the action of the levator palpebræ superioris by a strip cut from the centre of the tendon of the superior rectus muscle of the eyeball. This tongue of muscle he stitched into position between the tarsus and the orbicularis of the affected upper eyelid.

The only contra-indication to Motais' ingenious operation is furnished by those cases of congenital ptosis associated with defective movement of the eyeball upwards. Under such circumstances the superior rectus tendon is presumably absent or, at all events, so imperfectly developed as to be useless for the purposes of the operation. For some weeks after the operation the eyeball is directed downwards, so that if the patient be old enough and the sight be good, diplopia is complained of. This unpleasant feature, however, according to Haab ('Atlas and Epitome of Operative Ophthalmology,' 1905, p. 302), "has always disappeared after a period varying from two weeks to five months at the latest in all the operations of this kind that have so far been reported." On the other hand, it is only right to point out that in a case mentioned by Dr. Félix

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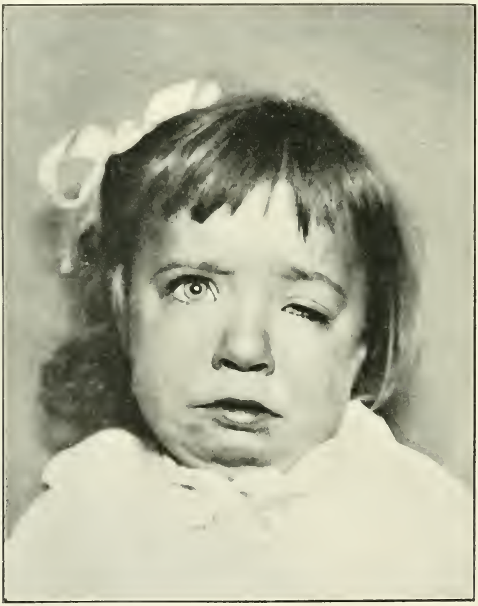
* 'Annales d'Oculistique,' June, 1897.

FIG. 3.



Terrien ('Chirurgie de l'œil et de ses Annexes,' 1902, p. 287) the diplopia had not disappeared six months after the intervention.

Although Motais' operation is by no means easy to perform even by one who is in the habit of dealing with the external ocular muscles, yet I have had almost perfect results in adults by following closely the inventor's original directions. In children, however, it becomes a matter of real difficulty to detach a central strip from the superior rectus muscle, which in young subjects is very small for the purpose. In the following case, therefore, I operated by dividing the superior rectus muscle lengthwise into an inner and an outer part, and by fastening the latter into the drooping upper eyelid through a buttonhole made through the conjunctiva. The result, as shown by the accompanying photographs, has been good.

The essential facts of the case follow:

Lily J— aged 2 years and 10 months, seen at the Evelina Hospital on September the 2nd, 1907. She was the youngest of nine children (four males and five females), none of whom showed any deformity of the eyes or other parts of the body. Family history unimportant. The left eye was the subject of incomplete ptosis, the pupil being practically covered by the drooping upper eyelid (see Fig. 3). No epicanthus. Upward movements of eyeball good. No synkinetic movements noted. No associated deformities. On September the 24th, under chloroform, the superior rectus was divided into two parts, the outer of which was stitched into the upper lid, and the knots tied on the skin surface of the latter. Seven days later the sutures were removed.

(*Exhibited October the* 18*th*, 1907.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said members would agree that transplanting the superior rectus to take the place of the levator palpebræ seemed a reasonable and sound operation. The superior recti were associated with the elevation of the upper lid and had the same nerve supply. His knowledge of ophthalmic surgery dated from so long ago that it would be of scarcely any service to relate his experience in the wards of Guy's Hospital. But he could remember cases of congenital ptosis. He had drawings of one, a youth with congenital ptosis of both eyes, who was able to see with both eyes only by throwing his head back. The operation performed was to cut an oval slice from the upper lids so as to shorten them. The result was not a perfect one, but it enabled the man to see without throwing back his head. In reply to Mr. Stephenson he said the chief reason the result was not brilliant was that they were very careful to preserve the power of closing the eyes during sleep, and perhaps were over-cautious in judging the amount of lid to be removed.

9. A CASE OF GENERALISED ALOPECIA.

By GEORGE PERNET, M.D.

This case had already been brought before the Society about two and a half years ago,* the scalp being then completely bald, and like a billiard-ball. Dr. Pernet had then pointed out how important it was not to give up such cases as incurable unless some years' treatment had been unavailing. The boy now showed a very respectable crop of pigmented hair about the head, some 1½ to 2 inches long. The treatment had been local stimulation, and internally thyroid. The patient, who presented the Mongoloid type of idiocy, had also improved mentally.

(*Exhibited October the* 18*th*, 1907.)

Discussion.

Dr. George Carpenter asked whether Dr. Pernet had tried carbolic acid spray to restore hair. Some years ago, when the Chairman, Mr. R. Clement Lucas, used to operate on tubercular hips at the Evelina Hospital, and when he, Dr. Carpenter, used to dress them and use the carbolic spray, and by the nature of the then fashionable operation they were very long in healing, he had noticed a considerable growth of hair in the neighbourhood of the wound. That led him to think that possibly such an application might be useful for the treatment of alopecia, and he therefore brought this fact to Dr. Pernet's notice.

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* See 'Reports,' vol. v, 1905, p. 230.

The Chairman (Mr. R. Clement Lucas) asked whether Dr. Pernet intended to operate on the defective eyebrows and eyelashes at present not evident; also had he adopted any local treatment? He could bear out what Dr. George Carpenter had just referred to, as to the growth of hair after the use of the carbolic spray in the dressing of wounds. But he thought it would be difficult to find such an apparatus at the present time. He supposed that any irritant would have the same tendency to promote the growth of hair.

Dr. Pernet, in reply, said he had no experience of the carbolic spray as a therapeutic measure for baldness.

10. A CASE OF SYNOSTOSIS OF THE UPPER ENDS OF THE RADIUS AND ULNA.

By HUGH LETT, Ch.B.

The patient is a girl aged 2 years. None of her relatives nor any of her nine brothers and sisters are similarly affected. Both arms are involved, and skiagrams show that the radius and ulna are united by bone for about an inch at their upper ends; the lower ends of the bones appear to be normal. The right radius is much curved. The right arm is in a position of nearly complete pronation, the left arm midway between pronation and supination. Flexion and extension of the elbow-joints are perfect.

(*Exhibited October the* 18*th*, 1907.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said that congenital ankylosis was very rare; that was the conclusion based on his own experience. Some explanation, such as intra-uterine fracture, seemed necessary to account for the condition.

Mr. Hugh Lett, in reply to the Chairman, said he regarded it as congenital. There was no history of anything of the kind in the family.

II. SIMULATION OF SOME OF THE SYMPTOMS OF PRIMARY AMAUROTIC IDIOCY BY A TUMOUR OF THE INTERPEDUNCULAR SPACE.

By EDWARD CECIL WILLIAMS, M.B.

THE subject of the following notes—an illegitimate child—aged 15 months, was sent under my care into the Bristol Children's Hospital by Dr. McClelland, of Brislington, with the following history. Three months ago she began to fail taking notice of objects, was losing flesh and getting weaker. On admission, the child lay quite still and motionless, was somnolent and not disturbed by any noise, even though close to her ears. Her eyes were firmly closed and when the eyelids were opened she took no notice of her surroundings. When raised up in bed her head rolled either backwards or to one side. The arms were slightly spastic, wrist-joint flexed. There were slow athetoid movements of fingers. The legs were spastic, and when the patient was elevated from the bed her legs remained extended. Knee-jerks and plantar reflexes were exaggerated on admission, but later on the spasticity was decreased and reflexes nearly disappeared. Babinski's sign was present; sensation was diminished. A slow reflex could be obtained by stimulating the skin of the opposite leg with a pin. There were sub-crepitant râles at the base of both lungs.

Examination of the eyes.—Pupils moderately dilated and fixed. Right divergent strabismus. No fundal changes, discs and macula both being normal. The child died nineteen days after admission from œdema of the lungs and heart failure.

Post-mortem examination.—Body much emaciated. Rigor mortis not well marked. Lungs congested and signs of bronchitis. All the other thoracic and abdominal organs appear to be healthy. Brain extremely soft. At the base of the brain is a fungating, cauliflower-like, yellowish growth, about the size of a walnut in the interpeduncular space. The attachment of the tumour to the brain-substance is most intimate at the anterior portion of the interpeduncular space, and is apparently a growth of the pituitary body.

The growth is well defined and presses upon: (1) the optic chiasma; (2) crura cerebri; (3) anterior portion of pons; (4) floor of the third ventricle.

There is a small yellowish growth situated on the right superior parietal convolution about one inch from the middle line. It is of the same nature and invades the grey matter of the cortex for about a quarter of an inch. The lateral ventricles are very much distended with fluid. Microscopical examination shows the growth to be a round-celled sarcoma.

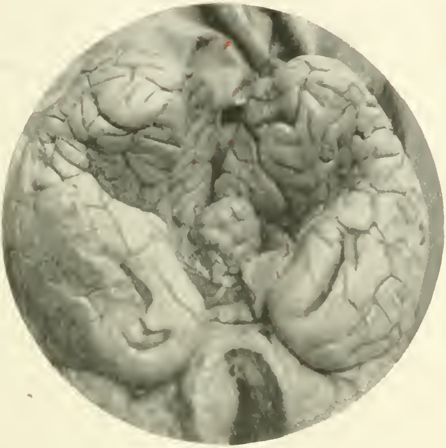


FIG. 4.—Photograph showing growth.

On admission it was thought possible that the case was one of primary amaurotic idiocy, as it exhibited several of the characteristic symptoms of that disease, viz.:

(1) Paresis or paralysis of the greater part of the body, which may be either flaccid or spastic.

(2) Reflexes may be normal, exaggerated or diminished.

(3) Diminution of vision leading to absolute blindness.

(4) Marasmus and a fatal termination.

The case did not, however, present the characteristic fundal changes, described in primary amaurotic idiocy, in the macula and disc. The child was not of Jewish origin. The progress of the case was more rapid than is usually the case in primary amaurotic idiocy, in which the symptoms usually begin earlier than in this case and the child lives longer. The symptoms in this case can probably be explained by the pressure exerted by the growth on the surrounding brain substance. I am obliged to Drs. Morris and Ridge, house-surgeons at the Bristol Children's Hospital, for the photograph of the brain and notes of the case, and to Dr. Fortescue-Brickdale for the microscopical examination of the growth.

(*Read April the* 19*th*, 1907.)

Discussion.

Dr. George Carpenter said he did not gather that any changes had been discovered in the eye by the narrator, and inasmuch as ocular changes were the characteristics of amaurotic idiocy, he wondered how the case could be held to resemble amaurotic idiocy.

Mr. Sydney Stephenson said it would be interesting to know why the child was blind. When Mr. Waren Tay showed at the Ophthalmological Society the first case of amaurotic idiocy, the characteristic changes were described. He thought the title of the paper somewhat faulty, and suggested that the Secretary should amend it a little. As there were no fundal changes the description did not seem to be a correct one. (Dr. Porter Parkinson pointed out that the title of the paper said "Some of the Symptoms.")

12. A CASE OF CONGENITAL HEART DISEASE IN A BOY, AGED FIVE YEARS.

By CHARLES W. CHAPMAN, M.D.

The only symptom this patient has is some shortness of breath on exertion. There is no history of his having had any illness beyond an occasional bronchial attack, and one so severe as to require confinement to the room for six months. Two years since he nearly died when under gas for the removal of tonsils and adenoids, and his condition was at that time so serious that the latter had to be left. The boy is exceptionally well-developed and his mental condition beyond the average of children of his age. The cardiac dulness is not increased. A thrill, systolic in time, can be felt in the pulmonic area, where also is heard a harsh systolic murmur conducted upwards and outwards; over the mid-sternum is heard a loud blowing systolic murmur, which can be traced in all directions and is faintly audible in the interscapular region; the aortic sounds are normal. The liver can just be felt below the costal margin; lungs normal. There is no cyanosis and the finger tips are not clubbed, but tapering. In all probability there is patency of the interventricular septum and stenosis of the pulmonic artery, both of congenital origin.

The interest of the case consists in the absence of the usual symptoms (with the exception of shortness of breath on much exertion), and in the unusually well-developed state of the child, who is the subject of marked cardiac lesions. (*Exhibited October the* 18*th*, 1907.)

Discussion.

Dr. George Carpenter said although there were no outward and visible signs in the case, he was quite in agreement with Dr. Chapman's diagnosis of congenital malformation of the heart and stenosis of the pulmonary artery. The child had a systolic murmur, best heard over the third left interspace close to the sternum, which was associated with a systolic thrill. The murmur, which was audible all over the front of the chest, was better heard on the child's left side than the right, and it was also to be heard in the great vessels of the neck. When the murmur was so conducted he looked upon it as a sign that the septum ventriculorum was perforate, but unfortunately it was not an infallible sign. A murmur, such as the one under discussion, indicated either stenosis of the pulmonary artery or a perforate septum ventriculorum, possibly one or other of these conditions, possibly both of them in combination; perhaps atresia of the pulmonary artery, or of the conus arteriosus, with a patent septum ventriculorum. He was not altogether in agreement with Dr. Chapman in regard to one point, viz. the absence of right-sided hypertrophy. He admitted that his examination was hurried, but he thought there was some hypertrophy of the right ventricle. He asked if Dr. Chapman based his opinion on a physical examination alone or by this combined with the use of the X rays. If the X rays were used he thought that question could be definitely disposed of. It was not uncommon for cases of congenital heart malformation to give no physical indications of their infirmity until an initial attack of lung trouble, in the shape of bronchitis or broncho-pneumonia, from the onset of which cyanosis and other signs often dated. Mild varieties of narrowing of the pulmonary artery, such as this appeared to be, were not incompatible with a long lease of life; indeed, Dr. Chapman's patient appeared to be singularly robust. The dangers lay in the balance of the circulation being upset by lung troubles and by the onset of endocarditis further complicating matters.

Dr. Edmund Cautley remarked that the patient was a good illustration of the fact that the prognosis in these cases depended on the general health of the child and the circulatory effects rather than on the loudness and distribution of the murmur.

13. A CASE OF RHEUMATIC HYPERPYREXIA IN A CHILD OF SIX.

By G. H. LOCK.

(Introduced.)

This rare and fatal complication of acute rheumatism hitherto, as far as I can discover, unrecorded in so young a patient, occurred recently in a girl, aged 6 years, whom I had attended at birth and since whenever she was ill.

Her father had worked in compressed air caissons for years and always successfully passed the periodical medical examinations to which such workers are subjected. Her mother was young and perfectly healthy. A maternal aunt had suffered from rheumatism, otherwise the family history was free from this disease and good in other respects.

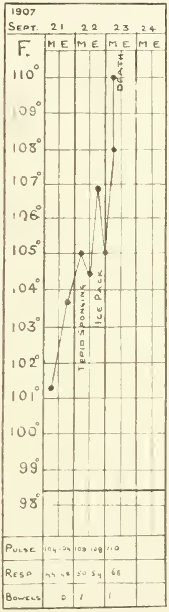
The child, with the exception of measles followed by whooping-cough, enjoyed good health during infancy. She grew up healthy, of alert intellect, and engaging disposition and manners.

A year ago she passed through an attack of chorea which subsided during six weeks' complete rest in bed, and when she was brought to me in January of this year to report progress she was fit and well, with the exception of a rough systolic murmur at the cardiac apex. I could not make out that the heart was appreciably enlarged, but the murmur was conducted into the axilla.

On September the 10th of this year she sickened for left lobar pneumonia which, with a temperature ranging between 102° and 103° F. and no uncommon features, terminated by crisis on the fifth day. A day or two after this crisis there was a return of the choreiform movements which, at first mild and confined to the left side, subsequently became general and severe.

On September the 20th pain in the left hip was complained of, and the temperature rose to 100° F. The child was enveloped in cotton-wool, and salicylate of sodium (5 gr.) in an alkaline mixture was given every four hours.

Fig. 5.



On the next day (September the 21st) other joints were involved, the temperature was 101° F., and the case was a typical one of acute rheumatism with chorea. The dose of salicylate of sodium was doubled.

The following day (September the 22nd) at 11 a.m., the temperature was 105° F. After tepid sponging the nurse reported a drop to 104·5° F. Salicylate of sodium (10 gr.) was administered every hour. At 6 p.m. the temperature had risen to 107° F. An ice-pack was ordered, and at 8.30 p.m. the temperature had fallen to 105° F. This application of cold was not well borne; alarming cyanosis and embarrassment of respiration followed. The heart distinctly dilated, and the sounds were almost inaudible. A drachm of brandy was given and ordered every hour throughout the night; the salicylate with the addition of 3 minims of tincture of digitalis was continued. Ice was kept applied to the head and nape of neck.

The next day (September the 23rd) at 10 a.m. the temperature was 108° F., pulse 110, respiration 68. A graduated bath was prepared, but, while waiting for a fresh supply of ice and before immersion, the temperature in less than an hour rose to 110° F. At this moment death occurred somewhat suddenly. An hour after death the temperature was 109° F. These temperatures were taken in the *rectum* by a trained nurse and verified by me. The last observation was kindly confirmed by Mr. S. H. Greene, of Kensington.

The onset of this period of abnormal temperature was not associated, as far as the chorea would allow me to observe, with any fresh endo- or pericarditis. The swelling of the joints was not so marked, but it was impossible to obtain anything from the child as to the degree of pain in them. The pulse, though feeble, was never greatly hurried, but the respirations were very frequent (60–68) and noisily panting in character. The most marked feature was the complete suppression of sweating. There was a plentiful crop of sudaminal vesicles from former sweats, but the skin was quite dry and pungently hot throughout.

There was no sleep during the presence of the hyperpyrexia; on the other hand there was no delirium or mania. The child, though dazed, asked for and knew her mother to the end, and the severe choreic movements ceased only with death.

I have searched through many writers for mention of this termination of cerebral rheumatism in childhood with negative results. That it must be extremely rare is evidenced by the remarks of Sir W. S. Church in his article on "Rheumatism" in Allbutt and Rolleston's 'System of Medicine,' 1906. In writing of hyperpyrexia, he states (vol. ii, part i, p. 625):

"Its occurrence in early childhood is as yet unknown," and, further, "no age period, if we except early childhood, appears exempt." In the same volume Dr. Cheadle, writing under the heading "Rheumatism in Children," states that he has never met with a case of hyperpyrexia, and that the youngest case of which he can find any record is that of a girl, aged 13 years.

(*Read November the* 15*th*, 1907.)

Discussion.

The Chairman (Dr. George Carpenter) thanked Mr. Lock in the name of the Society for his interesting communication, and said that he had many years ago seen a similar case to that now recorded, but he had not had sufficient leisure to search for the notes and give exact details, but he would find them and report to the Society later on.* Briefly, the child was younger than the present patient, a female some three or four years old. Hyperpyrexia occurred during an attack of chorea and with a fatal issue. At the time there was double optic neuritis, and prior to the hyperpyrexia rheumatic nodules, and antecedent to the chorea rheumatism. Hyperpyrexia was not confined to rheumatism. When resident medical officer at the Evelina Hospital for Children many years ago, he was in the habit of using the continuous hot wet pack in the treatment of acute renal disease, the patient being wrapped in a blanket wrung out of boiling water and surrounded by a mackintosh covered by a blanket. The temperature of the pack was about 130° F. If the temperature were not frequently taken during its administration, as a matter of precaution, there was risk of danger to life. One of the patients died, and he recorded the cases in the 'Practitioner.' †

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* Vide No. 14, p. 34.

† 'Practitioner,' September, 1888, No. 243, vol. xli, No. 3, "Dangers of the Continuous Hot Wet Pack."

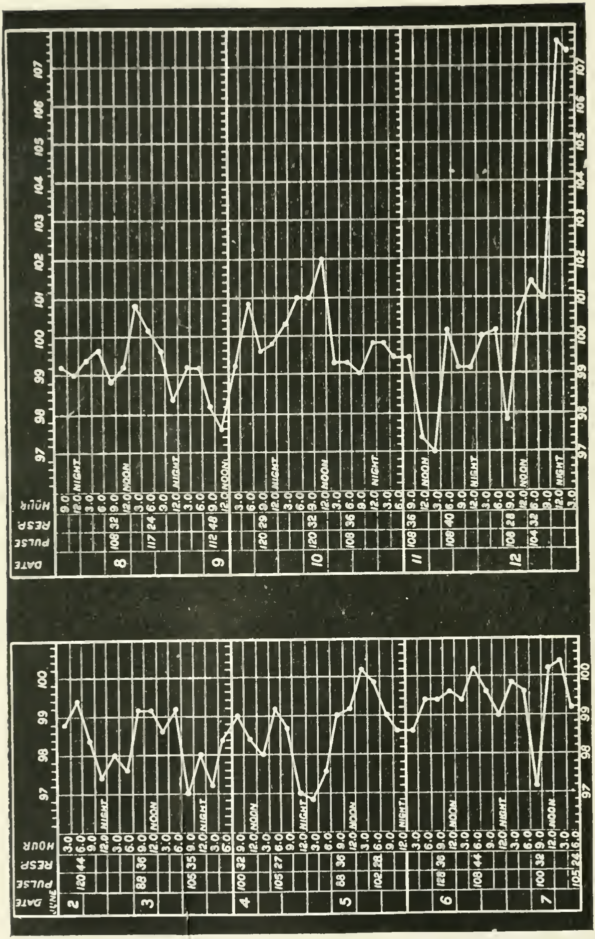

Fig. 6.—Chart I, Case 1 (first part).
Temperature taken in axilla.

Case 1 was that of a boy, aged 7 years, who was admitted into hospital with the objective signs of renal disease on June the 2nd. He had one third albumin; the pulse-rate was 88 to the minute, the respiration-rate 40 to the minute. He was ordered the continuous hot wet pack. On the seventh day he was suffering from pustular dermatitis, and on the twelfth day the temperature suddenly ran up to 107·6° F., over 6° F. between 9 and 12 midnight. From the time of his entry into hospital there had been some rise of temperature, but the highest previous record was 102° F. During the hyperpyrexia he was delirious, the face was pale, the lips parched, the tongue dry, the skin burning hot, and restlessness was extreme. He was taken out of the pack and sponged with water at a temperature of 90° F. After twenty minutes' sponging the body temperature had fallen to 104·2° F.,

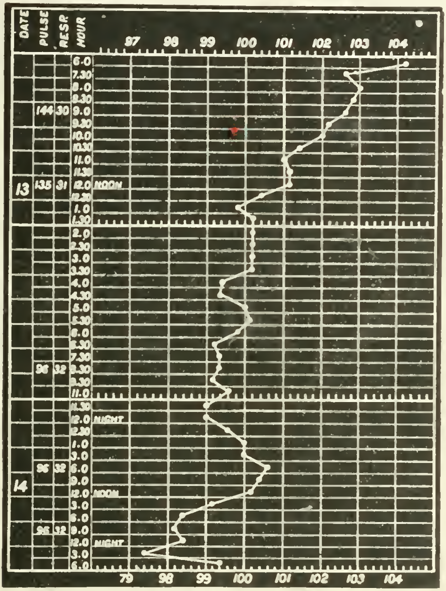

FIG. 7.—CHART II, CASE 1 (second part).
Temperature taken in axilla.

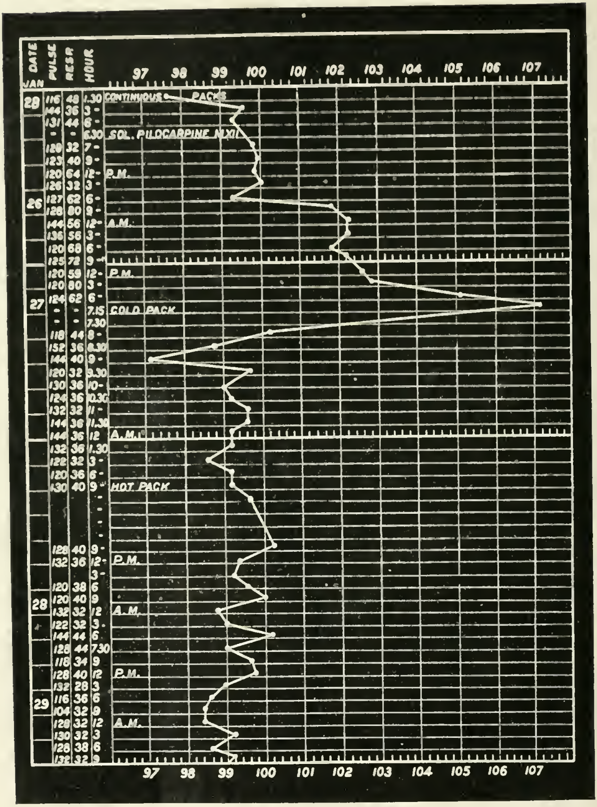

FIG. 8.—CHART III, CASE 2.
Temperature taken in axilla.

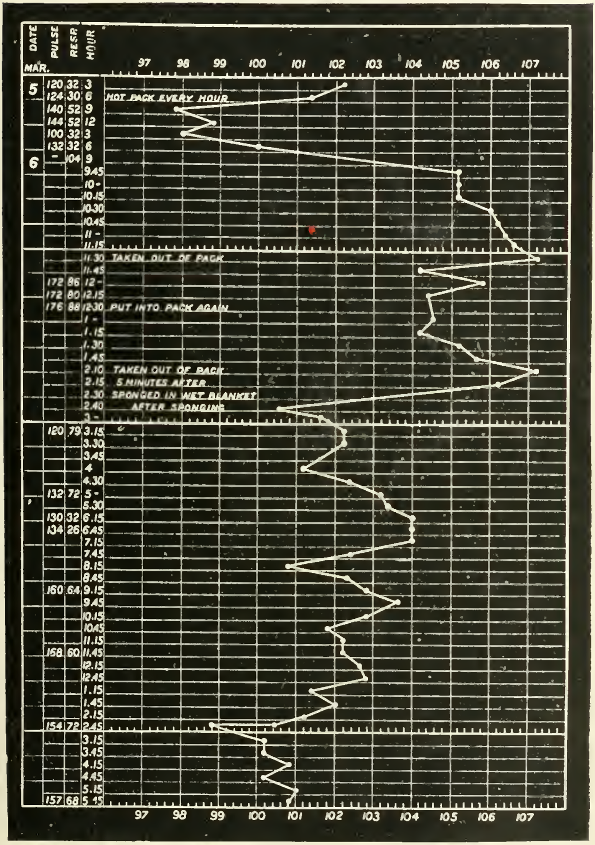

Fig. 9.—Chart IV, Case 3 (first part).
Temperature taken in axilla.

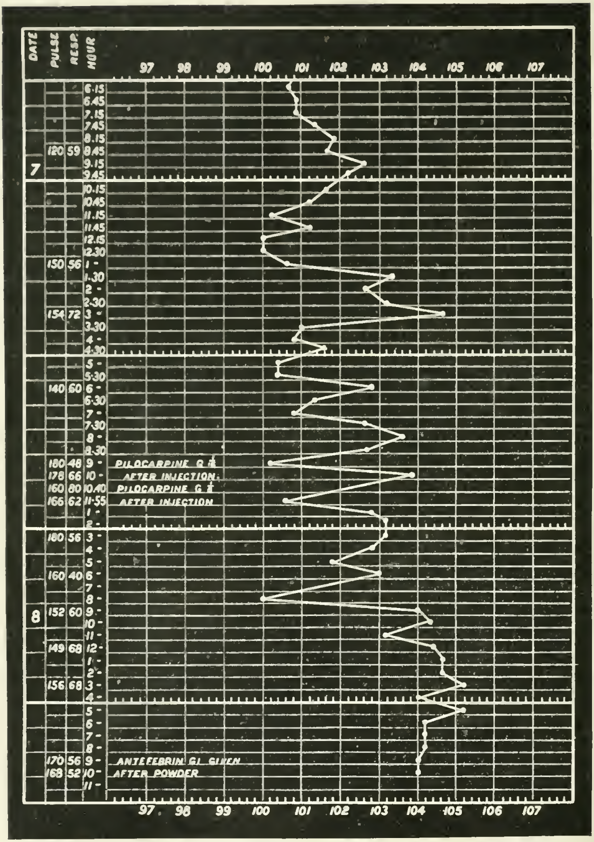

FIG. 10.—CHART V, CASE 3 (second part).

Temperature taken in axilla.

in one and a half hours after the commencement to 102·3° F., and it continued to fall hourly until it reached 99·8° F.

The next case was that of a boy, aged 3¼ years, who was admitted into hospital on January the 25th suffering from nephritis; the pulse-rate was 116 to the minute, the respiration-rate 40 to the minute, and the temperature subnormal. From the moment the continuous hot wet pack was started the temperature began to rise, and by midnight the following day it was 102·8° F. It then rapidly rose, and at 7 a.m. it was 107·2° F. He was very sick, the face was livid, the lips cyanosed, the respirations very rapid, the pulse barely to be counted, and he was tossing about his cot and delirious. He was sponged with water at a temperature of 95° F., which was cooled down to 60° F. Shortly after sponging commenced the delirium passed away and all urgent symptoms quickly disappeared. In half an hour the temperature had fallen to 100·2° F., and twenty minutes later it was all but normal, and at nine it was 97·2° F.

The third case was that of a girl, aged 2½ years, suffering from acute nephritis with a temperature of 102·2° F. The pulse-rate was 120 to the minute, and the respiration-rate 32 to the minute. The chest was resonant, but a few rales were audible back and front. She was ordered a continuous hot wet pack. Three hours after admission into hospital the temperature had fallen to 96·8° F. At 3 o'clock the following morning it was still subnormal, but from that time it rapidly began to rise until at 9 o'clock it was 105·2° F. Her face was then pale, her lips blue, and she was restless. By 11·30 the temperature was 107·2° F. The respirations were then very rapid and sniffing and she was very irritable. She was removed from the pack and wrapped in a dry blanket. A quarter of an hour after removal from the hot wet pack the temperature had fallen to 104·2° F. She was again replaced in the continuous hot wet pack and with the same result: the temperature, which at 12·30 was 104·4° F., had risen by 2·10 p.m. to 107·2° F. Convulsions then came on, she was more cyanosed, the face was ashen, the respirations very rapid, and the pulse at the wrist barely distinguishable. Previous to this she had been irritable, with periods of occasional delirium, and she was sick once. Judging her symptoms to be urgent, I gave her a cold douche from a water-jug which was close at hand and followed this by sponging. As she regained consciousness she began to gasp, and subsequently asked for water, and declined to take milk when it was handed to her. Her mother visited her at 6·45: she knew her quite well and talked to her. The respiration-rate was then 26 to the minute and the pulse-rate 134 to the minute, the temperature being 102° F. From that time she went downhill: the respiration-rate was increased (68), the face was pale, sickness supervened, she passed into a drowsy state, subsequently became very restless and delirious, and on the day following the onset of hyperpyrexia the physical signs of pneumonia were detected. She died at 11 p.m. that night; the temperature never rose above 105·2° F. At the autopsy there was general œdema of the lungs together with areas of red and grey hepatisation.

Two of the children sweated, one freely, the other slightly, right up to the hyperpyrexial stage. In one sweating had entirely ceased for many hours prior to the advent of hyperpyrexia.

Although some children may be placed in a continuous hot wet pack for days even without an untoward symptom, in others trouble is threatened immediately. Thus in one child it rose from 99° F. to 105° F. in twenty minutes, and in a subsequent experiment from 100° F. to 104·2° F. in three quarters of an hour. When using, therefore, the continuous hot wet pack it is necessary to keep an eye on the thermometer.

Dr. F. J. Poynton said that though very rare in children hyperpyrexia was not so rare in French literature. Three years ago he had a case in a child, aged 11 years, of which he published the full necropsy; it was remarkable firstly in that it obviously had an attack of rheumatism, but satisfactorily reacted to treatment. After a fortnight's convalescence, while still under treatment by salicylate, the child developed headache and fever and died. The temperature in the rectum was 106° F., whereas in the axilla it was never more than 102° F., as repeatedly verified. There was an organic lesion, namely, spinal and cerebral meningitis. At the discussion an attempt was made to show that it was not rheumatic, but without success; the organism was not the pneumococcus. That was the only case of hyperpyrexia in a child he had seen.

Dr. George A. Sutherland said his experience was that hyperpyrexia must be very rare in childhood; he had not met with a case. In 1882 the Clinical Society appointed a special committee to investigate the question of hyperpyrexia in rheumatic fever. They collected particulars of forty-seven cases, none of them being under ten years of age. He hoped Dr. Carpenter would find the notes of his case, because their inclusion would add to the value of the 'Reports.' In the Clinical Society's report the chief treatment referred to was by means of cold, and Mr. Lock seemed to have applied cold to his case, without benefit. He believed cold was not well tolerated by children with a high temperature; but there were some advocates of cold in the profession, such as Dr. D. B. Lees. Mr. Lock referred to the extreme dryness of the skin, and when that was present hot applications appeared to be indicated rather than cold.

Dr. C. O. Hawthorne said it was well to keep clear two classes of cases: one in which, as in the case just narrated, there was great elevation of temperature associated with some other active manifestation of rheumatism, such as chorea, and the other in which the high temperature appeared without any apparent organic explanation. In chorea, when there were such complications as endocarditis and pericarditis, the occurrence of a high temperature was not very uncommon, *i. e.* in severe and fatal cases. In adults, when speaking of hyperpyrexia, one generally meant a high temperature for which there was no obvious organic explanation. A marked feature of rheumatic hyperpyrexia in the ordinary use of the term was the cessation of all other active evidences of the disease. Therefore such cases as the present he would be disposed to call hyperpyrexia in rheumatic chorea, rather than merely rheumatic hyperpyrexia, as this latter term implied that the case exactly corresponded to the condition so named in adults. He had never seen a case of pure rheumatic hyperpyrexia in a child, but in view of the distinctly curative value of the cold bath when this condition occurred in adults, he would feel it impossible not to urge it in even a patient of tender years.

Dr. Frederick Taylor said the subject of the treatment was a very difficult one, because although the symptoms were sometimes removed by the use of cold, and therefore one might say the high temperature must have been the cause of the symptoms, yet in fatal cases where the temperature had been often reduced by cold there must be some other factor besides high temperature. It seemed likely that a toxin was operating in such cases, partly upon the cerebral centres, but probably also on other organs. Though he had not experience of that condition in children, it seemed obvious that cold should be brought into operation as soon as possible. Once, in an adult, he was very successful by rubbing pieces of ice over the surface of the body. The patient had a temperature of 107·5° F., and was comatose; there was no bath available, nor any means of immediately applying cold in any other way. After the use of the ice the patient became conscious and ultimately recovered. This treatment might not succeed in every case, and its use in a quite young child might have to be very carefully considered.

The Chairman (Dr. George Carpenter) said that in one of his cases of artificially induced hyperpyrexia the child had succumbed to pneumonia after application of cold, but whether it resulted from his action or not he was not prepared to say, but he thought it possible that it was so. Dr. Sutherland had spoken of the application of cold according to Dr. Lees' practice. He had found children bear cold well in the shape of the ice bag to the chest in cases of pneumonia. This application, in his experience, was tolerated, unless it was placed over the area of the heart. If placed there the child soon became collapsed.

Mr. G. H. Lock (introduced), in reply, said when the temperature was 105° F. tepid sponging was carried out, but its effect was only slight. The evening temperature being

107° F., an ice pack was applied, and that nearly killed the child, but its effect was to reduce the temperature 2°. The following morning the child died before he could apply the bath. If he had a similar case in future, which did not sweat, he would employ a hot pack.

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14. A FATAL CASE OF CHOREA ASSOCIATED WITH DOUBLE OPTIC NEURITIS AND HYPERPYREXIA IN A CHILD OF THREE AND A HALF YEARS.*

By GEORGE CARPENTER, M.D.

A FEMALE, aged $3\frac{1}{2}$ years, some six weeks before I saw her, which was in June, 1890, suffered from swollen hands and feet. Fourteen days after this attack she developed choreic movements which were general. When I saw her she spoke with difficulty and the movements were most marked in the face. They were much worse when she was being watched. She had a mitral bruit and there were numerous subcutaneous nodules the size of a bean on the back of the head and about the ankles and feet. The temperature was 100° F. A full diet was ordered, together with two minims of Fowler's arsenical solution three times daily to be increased by drop doses every other day. On July the 10th the arsenic mixture, which had reached 10 minim doses, was stopped because of conjunctival irritation and a discharge from the eyes. By August the 2nd she had wasted considerably and there was pityriasis tabescentium. The subcutaneous fibrous nodules had disappeared. The mitral bruit was then very harsh and distinct. There was double optic neuritis. She was taking her food badly and was languid and drowsy, but the choreic movements were less. Her morning temperature on this date, which had been previously subnormal, was found to be 101·6° F., and by midnight it had reached 106° F. She was quite conscious until 4 p.m. on the 2nd when she became delirious. The movements at this time were greatly augmented. The face was flushed and the skin hot and dry. A three-grain dose of antifebrin was given at 7 p.m., repeated at 9.40 p.m., and again at 12 midnight. Apart from the production of much sweating at the back of the neck the drug did not materially alter the condition. At 1 a.m. the following day the temperature had fallen a little more than two degrees, but quickly ran up to 106·4° F. at 2 a.m., when she was sponged, which reduced the temperature to 103·8° F. During this period the breathing was rapid, the face livid and the lips blue, and she swallowed with difficulty. She died at 4 in the morning.

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* 'Lancet,' November 30th, 1907.

Autopsy.—There was slight thickening of the mitral valve. Peyer's patches were injected but the intestines were otherwise quite normal. The brain appeared normal to the naked eye but no microscopical examination was made.

(*The case reported in brief to the Society November the* 15*th*, 1907.)

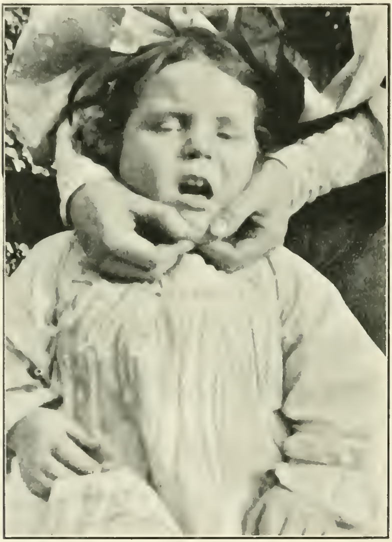
15. A CASE OF FRACTURED LOWER JAW.

By ELDON PRATT, M.D.

On the evening of July the 19th, 1907, I was called to a female child, aged 4 years, who had been picked up in a field with injuries to the face. It required very little examination to see that there was a compound fracture of the lower jaw; it was compound not only from the mucous membrane being torn, but also from the fact of a deep wound on the chin rather to the left side; the fracture was vertically through the symphysis, the two halves being freely movable on one another; in a position of rest, however, the left half of the jaw was at a higher level than the right half. One was able to get perfect apposition of the two halves by well opening the mouth, fixing the right half with finger and thumb, and then drawing downwards and slightly forwards the left half. There had been a good deal of hæmorrhage.

I cleaned and sutured the wound on the chin, and applied by means of bandage a roughly-made splint, placing a cork between left upper and lower jaws to keep open the mouth. In this way deformity was corrected and irrigation of the mouth was easy.

Fig. 11.



On seeing the child again next morning I expressed a desire to consult with a dental surgeon. Mr. Walter R. Wood, of Brighton, saw her with me that evening. He took a cast of the jaw and subsequently moulded a suitable splint. The splint here shown is the identical one used. In sending it me Mr. Wood reports as follows:

"You will notice that the gutta-percha has been partially scraped away, but will remember that before inserting the splint I filled up the hollow portion with gutta-percha, softened it over a lamp, and placed in position whilst warm. Apparently it kept perfectly in place the whole time the child wore it, and caused her practically no inconvenience, feeding, etc., being quite a simple matter.

"The two points of practical interest to me were:

"(1) The method for keeping the two halves in position whilst inserting the splint, *i.e.* the ligature round the left lower central incisor, on which one had to pull to get apposition.

"(2) The method of propping the mouth open, so as to allow of sufficient leverage with the bandage to keep the lower jaw well up in position. With an adult using an ordinary Hammond wire splint and four-tailed bandage one usually brings the jaws quite close together; but in this case it was impossible to keep the parts firmly in position without propping the mouth open."

I think the shape of the splint clearly speaks for itself.

The treatment proved very successful; when the splints were removed on August the 4th (fifteen days after the injury) union was complete.

The photograph here shown (Fig. 11) was taken on November the 5th, and clearly shows how perfect union was with complete absence of deformity.

The cause of the injury was doubtful, as there was no other witness. The child says that a horse kicked her. This I think probably correct, as there must have been sudden and strong direct violence to cause, besides the fracture, such a deep external wound on the chin.

In an adult fracture of the inferior maxilla through the symphysis is very rare. In a child fracture of any part of the inferior maxilla, therefore presumably much more so of the symphysis, is an exceedingly rare injury. I can find nothing about the subject in any book. The necessity of calling in the aid of a good dental surgeon is obvious whereby to carry out the necessary splint treatment, and to

Mr. Wood I am extremely indebted for his valuable help. Knowing how quickly fractures in children undergo repair, in a case of this sort it is important to have the splints made immediately to prevent deformity; union in this child was firm and bony at the end of fifteen days. One other point of importance is frequent irrigation of the mouth with a mild antiseptic.

(*Read November the 15th*, 1907.)

16. CASE OF VACCINIA.

By ELDON PRATT, M.D.

On September the 25th, 1907, I vaccinated a male baby when six days old in four places on the left arm. When seen on October the 1st all four places had taken well.

I was asked to see the child again on October the 11th as the vaccinated arm was very sore and the baby ill. On inspection I was indeed amazed to see a very sad state of affairs. There was extensive ulceration over an area measuring $3\frac{3}{4}$th inches by $4\frac{1}{4}$th inches, the ulcer being bathed in a purulent fluid which was discharging freely from the surface; the surrounding skin was very inflamed; and there were pocks of auto-inoculation near left axilla and over lower right ribs; the whole of the body, face and limbs was covered by a diffuse papular rash; tongue was coated, and the baby had frequently vomited, being quite off his feed. He was obviously suffering from *generalised vaccinia.*

I prescribed hot boracic lint fomentations to be applied to the large ulcer every two hours, and one grain of hydrargyrum c̄ cretâ in powder every night. The condition, both general and local, improved rapidly, and by October the 22nd the child was out of my hands.

A point in the family history is of interest. The mother has one other child, aged 4 years, which was also very badly affected when vaccinated. The mother herself when last vaccinated some six years ago "took" excessively. There was, therefore, I presume, an inherited predisposition to react powerfully to the vaccine.

I do not know whether this class of case is common. My experience of vaccination is by no means small, but I have not met with a similar case before. Such an extensive surface of ulceration formed by pocks becoming confluent seemed to me very unusual. There was no fault to find with the vaccine, as another child vaccinated with the same brand took only in two places. Dr. T. D. Acland, in his article on vaccinia ('Allbutt's System of Medicine') mentions a case of his in which a similar sore measured 4 by 4½ inches, eventually proving fatal. My case, with a sore nearly as large, and in a much younger child, happily proved otherwise.

(*Read November the* 15*th*, 1907.)

Discussion.

Dr. Charles W. Chapman asked whether there was anything wrong with the drains or whether the child's clothes were quite clean?

Dr. George Pernet said he had seen a case somewhat similar in which the vaccine pustules became confluent and formed a large ulceration with much brawny infiltration extending down the arm. It got well very slowly despite careful treatment. The papular eruption to which Dr. Pratt referred was the result of a toxin, which was brewed on the ulcerating surface. Such a rash was formerly called vaccine lichen. He wrote a paper some years ago on vaccination rashes and their complications, in which Dr. Pratt would find many details.* He had seen several cases of generalised vaccinia in which all the lesions went through the regular stages of vaccine lesions. He considered they were the result of inoculation from the original vesicles. Trousseau has pointed out that the vaccine papule could be reproduced by experimental inoculation with diminishing reaction up to the tenth day.† In Dr. Pratt's case the complication at the seat of the inoculation was perhaps the result of infection from the child's nails.

Dr. Frederick Taylor said he had not much to do with vaccination, but the Chairman (Dr. George Carpenter) might remember a case at the Evelina Hospital in which a fatal result ensued. Dr. Pratt had suggested that there was an undue inherited disposition in the patient to suffer from the vaccine virus, but, as Dr. Chapman suggested, many cases were attributed to external sepsis. He would be glad to hear the modern view on the subject. The case he had under his care at the Evelina Hospital, in regard to which he was required to give evidence before the Vaccination Commission, presented over the whole shoulder an infiltrated sloughing mass, and there were two or three vesico-pustules on the chest exactly like original vaccine vesicles. The vaccine poison in that case appeared to operate upon other parts than those at which it was originally introduced. The mother was in a comfortable position, and she had her own medical man, who said he had vaccinated the child most carefully with a clean needle; and another child had been vaccinated with the same lymph and was perfectly well. Possibly the chest was inoculated from the arm.

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* 'Lancet,' vol. i, 1903.

† Trousseau 'Clinique Médicale,' vol. i, 1865, p. 64.

The Chairman (Dr. George Carpenter) said that in the out-patient department of a children's hospital cases of vaccination not infrequently came under notice, and in his experience urticaria papulosa was a not infrequent sequel of vaccination. And in those predisposed to it urticaria vesiculosa and urticaria pustulosa also arose. Further, erythema multiforme was also occasionally seen. Instances of pustular dermatitis from pus inoculations were also not uncommon. He had also seen considerable œdema of the corresponding arm and hand, and even erysipelas. In isolated examples true vaccine inoculations occurred from the transference of the vaccine virus to other situations of the body—the face for instance. But in Dr. Pratt's case he thought organisms other than the vaccine virus were introduced into the wound at the time of vaccination, and that Dr. Pratt's case could be explained in that way. He vaccinated himself some years ago, and with disastrous local results, for his arm became as large as a leg, and he subsequently experienced three months' illness. Certainly he had protected himself from smallpox, for he subsequently saw many cases of smallpox; but although he had adopted precautions to ensure a clean vaccination operation he had evidently failed in his endeavours on that occasion, and he thought Dr. Pratt had been equally unfortunate. There was another contention—the vaccine virus might not have been free from septic organisms.

Dr. Eldon Pratt, in reply, said he could not agree that it was a septic case at all. The house was in a good sanitary condition, and the mother had a comfortable home. He was very particular about sterilising everything. He thought there was an inherited predisposition to react vigorously; the child was only six days old when it was done. The baby's brother, four years before, and the mother herself also reacted violently. Should re-vaccination be done in seven years' time he believed there would be the same result. Dr. Pernet did not seem to have grasped the fact that the case was described as generalised vaccinia, not from the fact of the papular rash, but that there were two lesions of auto-inoculation, one near the left axilla and the other over the right lower ribs, and each of them went through all the stages of a vaccine pustule. The papular rash had disappeared in twenty-four hours, and he did not think there was any sepsis. Ordinary fomentations were very successful.

17. A CASE OF INTENTION TREMOR IN A CHILD OF FIVE AND A HALF YEARS.

By L. A. CLUTTERBUCK, M.D.

She was the first child of her mother, who was 26 years old when the infant was born; labour tedious, finally completed instrumentally. A second child born 1½ years later, healthy. There is no evidence of specific disease.

The child never had convulsions. Backwardness in walking and talking were the first things noticed; she did not walk till three years old. She seems rather backward mentally.

There is coarse intention tremor of the hands; there are also marked inco-ordination and spasticity of lower limbs; no nystagmus, but lateral movements of the eyeballs are not smoothly made. Knee-jerks and plantar reflexes are increased; the response on plantar stimulation tends decidedly to be extensor. Talks slowly with separation of words and syllables, and almost entirely in a monotone.

There are no changes in the fundi.

(*Exhibited November the* 15*th*, 1907.)

Discussion.

Dr. Edmund Cautley thought it belonged to the great group of cerebral diplegias, resulting from either injury at birth or incomplete development at birth. From the history of difficult labour he gathered there had been either slight meningeal hæmorrhage or possibly encephalitis, and that the effects had fallen on the motor cells of the cortex. From a physical examination he thought the child was not quite as bright and intelligent as she should be at her age. He thought the prognosis in that kind of case was rather good—that she would improve considerably.

Dr. George A. Sutherland would hesitate to suggest disseminated sclerosis in a child, as the author had done, especially when the symptoms had apparently dated from birth. It was a very rare affection in early life. Possibly there was a cerebellar lesion, which would account for some of the symptoms of marked intention tremor. The mental backwardness was rather marked in this case, and there was spasticity of the limbs. He agreed with Dr. Cautley in thinking the original lesion was meningeal hæmorrhage. This had probably affected the cerebrum generally and possibly also the cerebellum. If it were simply a case of non-development or delayed development of the cerebellum there would not be spasticity. Dr. Cautley gave a hopeful prognosis, but he, Dr. Sutherland, would give a grave prognosis, as the child had been suffering from birth up to 5½ years of age. He thought the spasticity would tend to increase, and that the mental development would be delayed.

Dr. F. J. Poynton did not think the peculiar tremor was associated with either the cortex cerebri or the cerebellum, but with a lesion of the red nucleus. He agreed that it probably dated from birth and that there was probably some diplegia. He agreed with Dr. Sutherland's less hopeful prognosis rather than with Dr. Cautley's.

Dr. Cautley explained that his prognosis was not that the child would get well, but he thought that as the brain developed the child would learn to accommodate its muscular movements in a more orderly manner, and in that way it would improve.

Dr. F. Parkes Weber asked whether there were any signs of congenital syphilis in Dr. Clutterbuck's case. Cases had been published of what might be termed "syphilitic multiple sclerosis in children." True multiple sclerosis was very rare, if it occurred at all, in children.

Dr. L. A. Clutterbuck, in reply, said he only suggested disseminated sclerosis with much diffidence; he was inclined towards it being a cerebral lesion. Sahli spoke of definite ataxia and intention tremors due to lesions of the cortex. His own and the father's impression was that the case was improving; the child made better efforts to do things and was now walking better. There was no history of syphilis in the family, nor indication of it in the child. A second child, born a year or two later, was healthy and sound in every way. He would try to bring the case again later.

18. A CASE OF CEREBRAL TUMOUR.

By GEORGE CARPENTER, M.D.

Henry P—, aged 11 years, was admitted into the Queen's Hospital for Children under my care on September the 6th, 1907. He enjoyed good health until a month previous when he became languid and complained of headache, at first situated over the right eye but since then over the forehead. Attacks of headache came on twice a week and lasted for about an hour. The attack was accompanied by sickness and he vomited "white phlegm." The recumbent position relieved the attacks. For a fortnight prior to his admission into hospital the paroxysms had increased in intensity and for two days before the headache was very severe. He sought relief as an out-patient but nothing abnormal was detected, and he was transferred to the Eye Department, where vision was found to be $\frac{5}{18}$ for both eyes. Atropine drops were ordered and he was directed to attend the following week. But the headache became much worse, he was very constipated, and he was losing flesh, so he was admitted into the hospital.

When admitted he complained of severe frontal headache, he was somewhat collapsed and his pulse and respiration were remarkably slow—the former 48 beats to the minute, the latter 12. The knee-jerks were present. The fundus oculi was examined by the house-physician and pronounced "normal." The *state on admission* relates that "considerable error of refraction was found" in the Eye Department.

Twelve days later the house-physician found double optic neuritis. In the interval he had been sick, complained of headache, the pulse-rate varied from 60 to 66 per minute, the respiration-rate about 22; he took his food badly, was very constipated and the temperature was normal.

On the night of October the 13th he became unconscious for about three minutes, with twitching of the left arm and leg, and the notes described slight weakness of the external and internal recti muscles of the eyes. The optic neuritis was steadily progressing. The knee-jerks were not obtained. Headache was still severe at times but there had been no sickness for several days. He was taking his food fairly but was wasting rapidly. The pulse and respirations were still slow and the bowels confined.

October the 17th.—"There was noted, practically ophthalmoplegia externa of both eyes." Extreme limitation of ocular movements in all directions, but can screw up the lids and open the eyes quite well. The optic discs were swollen and enlarged in all directions, and presented striate hæmorrhages and exudations. The retinal veins were dark and tortuous, the retinal arteries if anything diminished in size.

The right yellow spot could be seen with a + 5 D. lens, the summit of the optic disc with a + 10 D., a swelling of + 5 D. or 1·5 mm.

The left yellow spot could be seen with a + 6 D., the summit of the optic disc with a + 8 D.; the swelling was, therefore, + 2 D. equalling 0·6 mm.

November the 15th.—Since October the 18th headache has been much less, and he has been sick only once since the 21st ult. But he now has almost continuous incontinence of urine and fæces. He lies on the side with all his limbs flexed, apparently taking little interest in his surroundings, though placid and happy. If disturbed he talks rationally, distinguishing between doctors and nurses by their voices, and answers questions about his health. He also calls out if he requires anything. He asks for the "bottle." When asked if he would like his tea he readily replied "Yes," and added, "and an egg too." Wasting is marked and weakness is increasing.

He is blind, and there is paralytic mydriasis and hippus in addition to other ocular troubles.

The knee-jerks are absent. The *right* plantar reflex is (?) flexor (plantar flexion), the left plantar reflex is (?)

dorsiflexion. The abdominal and cremasteric reflexes are present. Otherwise there are no physical signs. He has a large pigmented mole on the right thigh.

Soon after his admission into hospital Calmette's ophthalmo-reaction was tried with negative results. The day before yesterday (November the 13th) on a *second* trial a positive reaction was obtained.

A recent development has been tenderness of the scalp, especially of the occipital region. Twice the boy had undergone lumbar puncture and with negative results. The boy was brought to obtain an expression of opinion by the Society as to the advisability or not of trephining. The exhibitor thought trephining should not be done because it would leave the child with a hernia cerebri with its attendant risks; that the child, although blind was perfectly happy; that the prospect that the tumour would remain stationary was a remote one even if the operation were performed; and that the prospect of recovery of sight together with that of increased mental powers could not be considered of advantage to the boy seeing the hopelessness of the case. He thought the boy had a tuberculous tumour of the pons.

(*Exhibited November the 15th,* 1907.)

Discussion.

Dr. F. J. Poynton agreed that there should not be an operation. Even if one were done he thought the sight would not return, but atrophy now would almost certainly result. Moreover, such operations often were not as successful as was hoped, and there was a danger of hernia cerebri, the leaking from the wound, and finally meningitis. It was strange how long such children might remain in the state of unconsciousness. A patient of his, first seen three years ago, remained unconscious from Easter last to a week ago—over six months—with a subnormal temperature for three weeks before death.

Dr. Porter Parkinson said that though he was strongly in favour of trephining in most cases of cerebral tumour where there were head symptoms, the present boy was so comfortable that it would be better not to subject him to the danger of operation. Operation was sometimes of great benefit. Last session he showed a child who had had convulsions, was at one time blind, had intense optic neuritis, and suffered from severe headache, convulsions, and vomiting. After the operation the headache and vomiting ceased, the sight returned, and the condition of the optic disc got so near normal that the average observer might regard it as normal. The child was still alive, and the symptoms of tumour had disappeared, while there had been no more convulsions.

Dr. C. O. Hawthorne agreed that the case was not one for operation, and said his experience of operation in cases of cerebral tumour was not an encouraging one. It was desirable to have on record not only cases where success was attained, but those in which trephining proved unsuccessful or disastrous. He asked whether in this or other cases repeated lumbar puncture had been tried. Drawing off the fluid twice a week or so would surely reduce intra-cranial pressure, and might therefore be as beneficial as trephining. He had tried it in two cases, one of which got well except that she was blind from optic nerve atrophy. The other was still under treatment. He asked whether the Calmette preparation used was the same on the two occasions. He had had a similar experience in a cerebral case, probably one of tubercular meningitis. The fluid preparation was first used, and there was no reaction, and a week later the solid tuberculin dissolved *ex tempore*, when there was a very marked reaction.

Dr. Edmund Cautley said that last February he showed a boy before the Society as a case of cerebral tumour. There was very marked double optic neuritis and paralysis of one external rectus, and he asked for opinions as to whether the case should be trephined. What he heard encouraged him to have it done, but the parents would not consent. The boy was now well except for the persistence of the double optic neuritis. No doubt some cerebral tumours became quiescent. In the present case it would be futile to operate. It was questionable whether improvement could be expected as the result of trephining in optic neuritis unassociated with other signs of increased intracranial tension.

Dr. Porter Parkinson said that in his case of cerebral tumour he tried lumbar puncture on two occasions, but could not get anything out either time. After that the operation of decompression was done.

Dr. George Carpenter, in reply, said his experience had been similar to Dr. Parkinson's, for he had twice attempted lumbar puncture, but with a negative result. With regard to Calmette's ophthalmo-reaction, the fluid tuberculin test was used, the preparation had been obtained from the usual source, and there was no reason to suppose there was anything wrong with it. He knew instances where this test had been used in obviously tuberculous cases, but the reaction had not occurred, whereas on a second trial it had, and it had been proved to be correct post mortem. The assumption, therefore, in the present case was that the tumour was tuberculous. He remembered a small child many years ago with slow pulse and double optic neuritis, but who developed no other symptoms for months. The patient went to a convalescent home twice, and finally returned to the hospital and died of tuberculous meningitis. A small tuberculous tumour was found at the autopsy in the cortex cerebri, and he presumed that the optic neuritis was secondary to that; the meningitis was certainly secondary to the tuberculous tumour. Tubercular tumours in the eye often grew very slowly, and he inferred from his observation on the fundus oculi that they could be of equally slow growth in parts of the body that could not be seen. He thought the illustration he had given would explain Dr. Cautley's case. He was glad members agreed with him as to the futility of operation in the case he had shown.

Postscript, August, 1908.

December the 4th.—The boy thinks he can see "lights." Optic discs still choked. Quite comfortable, no headache. Since the last note has been sick three times. On December the 5th tuberculin injections were commenced (tuberculin R $\frac{1}{2000}$ mg.), and five injections were made between that date and December the 23rd; the temperature throughout was subnormal, and on only one occasion did it reach 99° F.—the evening that an injection was made. Between these dates he was sick ten times. His pulse-rate varied between 72 and 96 to the minute, the respiration-rate between 20 and 28 to the minute. On December the 9th it was found that both discs were becoming pale—distinctly white, the right side especially. There were then two areas noticeable, an inner pallid part and an outer ring of haze, a halo, as it were, at the outer edge of which the blood-vessels could be seen to bend over. His mental condition had improved; he readily answered and asked questions, *e.g.* "Shall I be able to see before Christmas?" and was not so lethargic. He took food well and enjoyed it, but the wasting continued. But on December the 16th he was more apathetic and drowsy. His pulse was irregular as regards time and ventricular force and was smaller. However, on Christmas

Day he was very lively and excited, singing and talking, when he suddenly collapsed and was found unconscious by the nurse. His eyes were fixed and staring and he had a frightened look in his face. This condition lasted for about five minutes, then passed off, when he complained of pains all over his head. After that he soon became himself again and was cheerful during the rest of the day. The pulse did not alter. The tenderness of the head previously noted when it was brushed still continued. By December the 30th he was weaker and more drowsy, and had complained of headache the previous few days, and on January the 6th, 1908, there was some little difficulty in rousing the child, who only answered questions after an interval of a few seconds. Constipation was still a feature, and he had been sick fifteen times in a month. On January the 16th he was unconscious and lay in any position he was placed, and passed his motions under him and was still occasionally sick. There were still signs of optic papillitis and nothing like white atrophy to be seen. The discs were pale but the vessels still obviously bent as they crossed their edge, and there was exudation on some of them in their immediate neighbourhood. The arteries were attenuated, the veins of natural size, tortuous. When the examination of the fundus was concluded he roused up a little and asked, " Who is it ? " in a sleepy way. On January the 21st the following note was made : " He lies torpid with closed eyes, pupils dilated ; does not see or take notice ; left eye less closed than right ; right eye more prominent than left ; right angle of mouth drawn up, and the left side of the face seems more placid than the right. Neither knee-jerks, ankle clonus nor Babinsky obtained, but the right plantar reflex seems inclined to be of the flexor type. Lies with the legs drawn up and neck seems rather rigid. Still, as heretofore, subnormal temperatures."

On February the 20th there was a note to the effect that there had been no striking change. He occasionally complained of pain in his side, which could not be definitely localised and which appeared to be due to position—

it changed from side to side. He sometimes complained of headache and pain at the back of the neck. He was almost deaf, blind, cried out aimlessly periodically and when touched. He always had his legs drawn up and the knee-jerks were not obtained. Slight extensor plantar reflex, not definite. Lately the head retraction had been more marked. He was sick during the first week of February but not since. During the preceding month the pulse-rate had varied between 60 and 96 beats to the minute, the respiration-rate between 16 and 24, and the temperature had kept subnormal. He was still constipated; there was no albumin in his urine.

On February the 24th the left optic disc was decidedly pale—atrophic pallor—and it was ever so much smaller than it had been. The right optic disc was pale in comparison with what it had been, but it was still large and the vessels at the upper segment bent as they crossed the disc margin; thus there was still neuritis present in this eye.

In March there was no change. It was impossible to obtain any assistance from him in regard to a physical examination, and rigidities of his lower extremities prevented any observations on his knee-jerks, etc. During this month he was sick only once.

In April he was a mere log and was only just alive. On the 27th his temperature rose a trifle—99·2° F.; on the 29th and 30th it was a fraction higher. On the 30th he was sick once, the first time since March the 17th. He had been getting feebler and feebler day by day, and on the night of the 30th he died. There were occasions during the latter part of April when his temperature had sunk as low as 96° F. and when his pulse could not be detected, but in spite of that he managed to drag through several days in this state feebly hovering between life and death.

Autopsy, May the 1st.—Body very emaciated.

Lungs.—Slightly emphysematous.

Heart.—Walls rather friable. The pericardium contained about ʒij of fluid.

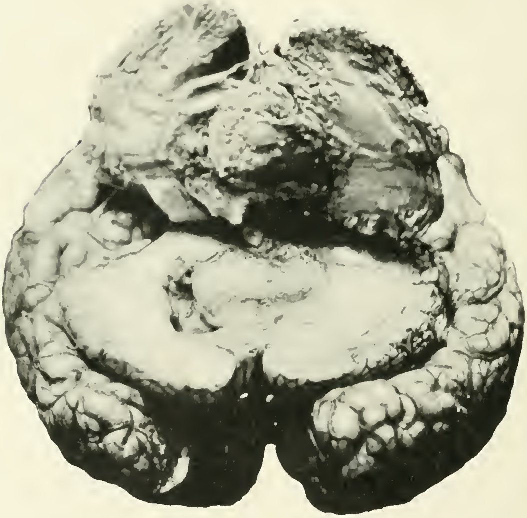
Liver.—Evidence of decomposition having set in.

Spleen.—Slightly enlarged.

Kidney.—Congested.

Brain.—On removing the skull-cap the brain was found to be bulging, and there was some engorgement of the superficial veins. The membranes presented a dull lustreless appearance, but nowhere were tubercles seen. On cutting the brain no lesion was found except some distension of lateral ventricles. On cutting the cerebellum a large tumour was found, extending from left to right across the middle line, and measuring 5 cm. it its longest diameter and 3·5 cm. in its broadest diameter (Fig. 12).

FIG. 12.



The left half of tumour showed evidence of breaking down and there was a cavity measuring 1·5 cm. in its longest diameter. The rest of the tumour was firm and of a reddish colour.

Microscopical examination by Dr. W. A. Milligan.—The section presents the appearance of a mass of round and spindle-shaped cells joined here and there by fibrous material. There is no evidence of caseation nor can any giant cells be found. Here and there are well-marked blood-vessels. *Conclusion:* Taking everything into account the favour of evidence, in my opinion, is that the tumour is of a sarcomatous nature.

The specimen was also submitted to Dr. Theodore Fisher, who was of the same opinion. I, too, am in agreement with these conclusions.

19. CASE OF ŒDEMA PERSISTING SINCE BIRTH.

By F. J. POYNTON, M.D.

Child aged 2 years, male. Swelling of the feet has been noticed since birth, varying in amount. Family history no bearing. The eyelids have been swollen in the mornings. There is now well-marked swelling of the dorsum of each foot, fading away gradually at the ankles and involving the toes; there is slight pitting on hard pressure. The feet are warm and of natural colour. By X rays the bones appear normal, the soft tissues being increased.

There is now no swelling of the eyelids. The urine is normal in amount, shows a faint trace of albumin and a few degenerated epithelial cells and scanty leucocytes; no casts or crystals. From a catheter specimen a Gram-negative motile bacillus was isolated and has been grown by Dr. Forbes. This resembles in its reactions *B. proteus vulgaris.* The child is well nourished, shows no abnormal signs in the chest or abdomen, and has had no digestive troubles.

Treatment with theolin-sodium acetate and digitalis was quite ineffectual.

I incline to the view that there is some renal fault of congenital origin.

(*Exhibited November the* 15*th*, 1907.)

20. A CASE OF ŒDEMA PERSISTING SINCE BIRTH.

By G. A. SUTHERLAND, M.D.

L. J—, female, aged 7 months, has been the subject of œdema of the feet since birth. The condition varies only in the degree of the œdema, not in the character. Under ordinary circumstances there is seen to be on each foot marked swelling of the dorsum, the sole, and the toes. The œdema ends abruptly at the ankle, which is freely movable. The dorsal swelling is rather pale and pits on pressure. The sole is tense and swollen, shows no pitting, and is of a pinkish-red colour. This colour disappears entirely on pressure. The toes are also pinkish-red, and are tense, and the nails are partially covered by the swelling around them. When the feet are cold they rapidly become blue or blue-black in colour. When the child is ill in any way the swelling of the feet becomes more pronounced.

The mother died of phthisis soon after the infant was born. A full family history cannot be obtained. The infant has suffered from gastro-intestinal disturbance accompanied by lichen urticatus. The striking feature about the latter was the appearance on the head, at the age of three months, of large urticarial wheals. These were as large as hazel-nuts, very tense, and white. Typical eruptions of lichen urticatus have appeared about the body and extremities at intervals.

On physical examination no cause for the œdema can be detected. The heart appears to be normal. The urine has been examined on three occasions. On two occasions there was present uric acid but no other abnormality. On the third occasion pus cells and a trace of albumin were detected, the latter being sufficiently accounted for by the former. The hands tend to get blue if at all cold. The eyes are at times noticed to be slightly puffy about the lids, the upper lids being as markedly affected as the lower. The foster-mother states that when the rash improves the feet are more swollen, and when the swelling of the feet diminishes the rash again comes back. The rash referred to is the lichen urticatus.

The nature of this form of œdema is obscure, but probably one may exclude cardiac or renal disease as the source. The blood has not been examined, but there is no definite anæmia. The œdema does not pass off under prolonged rest in the horizontal position. Possibly the association with lichen urticatus lends strength to the suggestion that the œdema is due to a general vaso-motor disturbance, intermittent in its action generally, but constant as regards the circulation in the feet. The outbreaks of lichen urticatus, the blueness of the hands, and the occasional puffiness of the lids show a tendency to disturbance of the peripheral vaso-motor system.

(*Exhibited November the* 15*th*, 1907.)

Discussion.

Dr. F. Parkes Weber regretted that he had not seen the patients, but Dr. Sutherland's description reminded him very much of the symmetrical condition sometimes referred to as "acrocyanosis in young adults"; in that condition the slightest cold caused cyanosis of the hands, sometimes accompanied by a certain amount of actual swelling. If the swelling in the child's feet were really analogous, it might diminish with improvement of the general condition.*

Dr. T. R. Whipham said he came to learn something about the condition. He had no explanation to offer, but could relate another case which was very similar under his own care. He did not know whether the condition existed from birth, but apparently it had been noticed soon after. Both feet and the left hand were affected with a hard, tense œdema, and there was urticaria completely round the trunk, which varied in extent from time to time but did not disappear. The liver was considerably enlarged. The patient had recently died from bronchitis, and he had not yet learned what was the pathological report.

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* On the following day Dr. Poynton kindly gave Dr. Weber an opportunity of seeing his case. Dr. Weber now believes the hard white œdema of the feet to be a form of "congenital trophœdema." Such a condition is said sometimes to be symmetrical, and there may be no history forthcoming of any similar trouble in the family.

Dr. Edmund Cautley said that in all such cases it was important to ascertain whether there was any family history of the condition, because there were hereditary cases of œdema of the lower limbs very much of the same type. Milroy reported that twenty-two out of ninety-seven members of one family in six generations were found to have suffered from it, and in every case but one it was congenital. It was not known whether the condition was due to blood stasis, or to a deficiency in the circulatory system, or lymph stasis, or absence of valves in the veins of the legs. Apart from the hereditary group, it was necessary to exclude cases depending upon renal inadequacy. In Dr. Poynton's patient it was suggestive that there were traces of albumin and renal cells, and it was possible there might be some renal inadequacy dependent on congenital malformation or on disease of the kidneys. Dr. Sutherland's case did not appear to have been thoroughly examined from that point of view. He believed the cases mentioned by Dr. Parkes Weber belonged to the cyclical albuminuria class. The dusky or bluish extremities were probably vaso-motor in their mode of production.

Dr. F. J. Poynton, in reply, said he agreed with Dr. Cautley's view of his case. He thought the fact that there was slight œdema of the eyelid, the aspect of the child before he saw it in the hospital, and the fact that when the swelling in the feet was greatest the urine was less, pointed to renal inadequacy. He wondered whether any congenital specific taint had produced the condition, but there was nothing in the family history to show it. He hoped to show the child again on a future occasion.

21. ABNORMAL DEVELOPMENT IN A GIRL.

By RUSSELL HOWARD, M.S.

Lilian U—, aged 7 years.

The patient is the child of parents of average weight, and there are two other children who are in no way abnormal.

The patient is alleged to have been a normal child till the age of 4 years. A great uncle of the patient, who is between 60 and 70 years of age, weighs 20 st., an aunt aged 31 years weighs 15 st., and an uncle aged 40 years weighs 17 st.

The patient is 3 feet 9½ inches in height and weighs 6 st. 4 lbs. The fat is evenly distributed about the body; the mammæ are well developed and the abdomen is pendulous, the whole appearance of the patient being that of a fat middle-aged woman.

The genitals are infantile in appearance and there is no growth of pubic hair.

The patient intellectually is childish and the expression of the face stupid. She is less developed mentally than the average girl of her age; for example, she cries if other children in the ward cry and she is not quite clean in her habits. She has never menstruated nor shown any abnormal sexual tendencies.

The heart and lungs appear to be normal, the urine is normal; no abdominal tumour can be made out; the thyroid is not enlarged and there is no evidence of enlargement of the thymus. Beyond suffering from enlarged tonsils and adenoid growths there is no evidence of any abnormal condition beyond the obesity.

Physical precocity in children in relation to the ductless and accessory genital glands was fully discussed by Dr. Leonard Guthrie at the British Medical Association of this year, and it would be waste of time to discuss any of the points which he then brought forward. The case I am showing to-day seems to fall into his classification under the heading of precocious obesity.

This case differs from the usual individual of this type in being somewhat feeble-minded, the type usually being mentally acute, although a number of patients showing feeble-mindedness have been recorded. The absence of pubic hair is also unusual, but the infantile condition of the external sexual organs with absence of precocious sexual instincts is the rule.

The absence of any evidence of physical disease, although, of course, it may easily be present and undetected, the history of obesity in this family and the absence of precocious sexuality, lead me to suggest that the case is one of simple obesity in which there is no abnormality of the ductless glands such as was described by Dr. Guthrie in the Clinical Society's 'Transactions' of 1906.

Before sitting down I should like to ask Mr. Lett if he can give us any particulars as to the further history of his case of precocious development in a boy with precocious sexual instincts. In his case there were nocturnal emissions, and I should like to know if living spermatozoa have ever been found in the semen.

(*Exhibited November the* 15*th*, 1907.)

Discussion.

Dr. F. Parkes Weber said he felt sure the case had no connection with what were ordinarily regarded as cases of "precocious development." There was nothing particular about this child except defective mental development associated with obesity. Bourneville, in France, had described somewhat similar cases.

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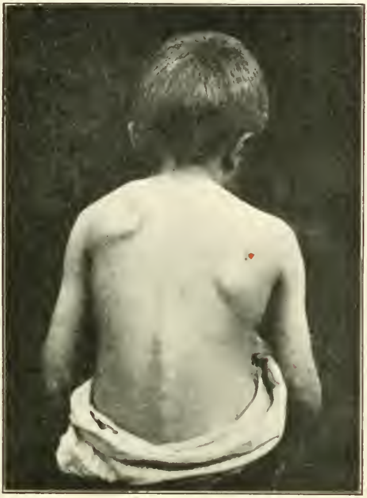
22. PHOTOGRAPH OF A CASE OF SPRENGEL'S DEFORMITY.

By RICHARD WARREN, M.Ch.

The photograph exhibited was that of a Jewish boy, aged 6 years. (See Fig. 13, p. 57.) There was shortening and fibrosis of the left trapezius. The scapula was displaced upwards, and there was bulging mesially of the vertebral border, which, however, did not articulate with the vertebral spines. (*Exhibited November the* 15*th*, 1907.)

FIG. 13.



23. A CASE OF CONGENITAL DISLOCATION OF THE HIP-JOINT.

BY RICHARD WARREN, M.CH.

THE patient shown was a girl, aged 5 years, and the unusual features of the case were that the neck of the femur was very small, and that there was no head to it.

(*Exhibited November the* 15*th*, 1907.)

Discussion.

Mr. J. Russell Howard said that the skiagram appeared to him to show a very unusual picture of congenital dislocation of the hip. As regards the success of Lorenz's method of treatment, he had treated a case of double congenital dislocation by this method with the following result. The right hip was first reduced with success, and then the left hip was treated. Unfortunately during the treatment the child developed measles, and the plaster had to be removed and treatment stopped. The result was a complete failure in consequence. At the present time the right hip is properly in place, and appears so normal that the case without the history would be considered one of left unilateral congenital dislocation. He advised that Lorenz's treatment should be carried out in the case shown.

Mr. Richard Warren, in reply, said he had not had great experience in the treatment of such cases, but he had seen good results in the hands of Mr. Openshaw by such method of reduction, and it was worth trying.

24. FACIAL PARALYSIS DUE TO ENCEPHALITIS.

By PORTER PARKINSON, M.D.

Alice H—, aged 3½ years, with a good family history, had had no previous illness. In the third week of September complained of headache and went to sleep and looked "funny" about the eyes. She was very drowsy for the next five days; weakness of the left leg was then noticed and her mouth went to one side when she laughed. She was admitted into the Queen's Hospital for Children a fortnight later. There was well-marked paralysis of the left side of the face involving the upper as well as the lower part; she was not able to shut the left eye or wrinkle the left side of the forehead; the left leg was weak but not paralysed, the muscles were somewhat rigid; the knee-jerk and the plantar reflex were not obtainable on the left side, but were normal on the right. The left arm seemed unaffected. The fundi of the eyes were normal. The muscles of the face gave the reactions of degeneration. In the left leg the tibialis anticus and the peroneus brevis reacted to faradism, but the remaining muscles of the lower extremity did not respond to either current.

It would appear that in this case there must be a double lesion, one involving the nucleus of the facial nerve, and also one affecting the motor cells of the anterior horn of the spinal cord in its lower part, so that there is a combination of encephalitis and anterior poliomyelitis.

(*Exhibited November the* 15*th*, 1907.)

Discussion.

Dr. F. J. Poynton said he would like to quote a case in support of Dr. Parkinson's view. He showed a case of double facial paralysis at the Medical Society of London as a case of probable fracture of the base, because the child had fallen down on his head. But there followed wasting of the leg, which had not been detected. He had no doubt now the child had double facial palsy, due to polio-encephalitis inferior, and had also infantile paralysis, and that there had never been fracture of the base. The fall was the result probably, and not the cause of, the sudden illness.

25. A CASE OF ACUTE ARTHRITIS OF THE HIP-JOINT; OPERATION; RECOVERY WITH PERFECT MOVEMENT IN THE JOINT.

By HUGH LETT, Ch.B.

Thomas G. R—, aged 11 years, admitted into hospital October the 30th, 1906. He had complained of pain in the leg for three days. No history of injury.

On admission there were sordes on the lips, temperature 102° F., pulse 110 to the minute. The left hip was flexed, everted and abducted, and any attempt at movement caused considerable pain. There was some fulness over the joint. The joint was at once explored by an anterior incision, and a small amount of pus was found in the joint cavity. A counter-opening was made posteriorly, the joint was thoroughly irrigated with saline solution, and tubes were inserted, a large one posteriorly and a smaller one anteriorly.

The temperature gradually subsided and was practically normal at the end of ten days.

The patient was discharged at the end of eight weeks with very good movement in the joint.

(*Exhibited November the 15th*, 1907.)

Discussion.

Dr. Edmund Cautley asked whether Mr. Lett knew what was the organism. Judging by the history and the duration of the case it seemed like a pneumococcal arthritis.

Mr. Hugh Lett, in reply, said that unfortunately no bacteriological examination was made, but he agreed that the infection was probably pneumococcal. There was no obvious disease of the bone and very little thickening of the synovial membrane.

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26. A CASE OF CONGENITAL CONSTRICTION OF THE RIGHT THIGH.

By A. H. TUBBY, M.S.

John M—, aged 1 year and 9 months, was brought to me at the Royal National Orthopædic Hospital in October, 1907. He is the fourth baby and is a healthy-looking child. There is a deep groove, ½ inch deep, which extends 1 inch from the left side of the median line of the sacrum, and passes across it to the right side, and then below the crest of the ilium. It is continued around it to just beneath the anterior superior spine, and then enters the fold of the groin and terminates at the outer side of the scrotum on the right side. Throughout the length of the groove distinct evidences of scarring can be seen. In association with this condition there is congenital talipes equino-varus of the right foot.

The question arises whether the equino-varus is due to nerve pressure or to mal-position *in utero*, but there is not sufficient evidence to decide, although the presumption is that mal-position is the cause.

But so far as the origin of the constriction is concerned there can be no doubt that it is due to pressure of a tight constricting band, and at the developmental age when this must have happened the only structure of importance which could have caused it is the umbilical cord.

While the origin of these grooves in the limbs is still a matter of supposition, it may be briefly said that when they arise early in fœtal life before the fourth month they are due either to amniotic bands or to the umbilical cord; in late fœtal life the majority of the cases arise from constriction of the umbilical cord. In some cases amputation takes place and the evidences of this are found when carefully looked for by the discovery of the amputated parts in the discharges at birth. If the size of the amputated part be great, or if the accident occur early in fœtal life, it is highly probable that death of the fœtus ensues. When the constriction is not such as to cut off the blood supply of the part the fœtus survives the whole term but is born with a constriction. In this connection Dr. Hawthorne's case, reported in the 'Reports of The Society for the Study of Disease in Children," vol. iii, p. 29, may be consulted.

(*Exhibited November the* 15*th*, 1907.)

27. LYMPHADENOMA IN A BOY, AGED SIX YEARS.

By PORTER PARKINSON, M.D.

This boy came from a large and healthy family. He had measles and diphtheria two years ago, and during the past six months the patient's mother has noticed lumps in the neck, but no other symptoms. The child is now anæmic.

The mass of glands is extensive, reaching from the lobule of the ear to the clavicle on the left side of the neck. Most of the glands are as large as a small walnut and very soft and elastic, but not fluctuating; they are freely movable over each other and the skin over them is normal. There is no pain. The veins in the neighbourhood over the arm and pectoral muscle are enlarged. There is no evidence of any other enlarged gland externally or in the chest or abdomen, and the spleen is not palpable. The temperature is normal, and so is the urine. The blood-count gives red blood-corpuscles 3,225,000 per c.mm., leucocytes 6688 per c.mm.; colour index 0·8.

(*Exhibited November the 15th,* 1907.)

28. "INHERITED SYPHILIS."

By R. CLEMENT LUCAS, B.S.

I HAVE had the honour of opening this discussion thrust upon me by the unanimous vote of the Council as a penalty for certain criticisms I ventured to make on former discussions, to the effect that the length and learning displayed in the opening addresses, sometimes five or six in number, left neither time nor opportunity for subsequent debate.

I ventured to think the opening should be more suggestive than dogmatic, and terse rather than discursive. As a consequence I am here to represent a phrase rather than a faculty, and the burthen of presenting new ideas and new facts will rest almost entirely with those who follow.

To begin with, this discussion will open under quite different circumstances to what it would have done had it been held three or even two years ago, for it is now generally accepted that the *Spirochæta pallida* discovered by Schaudinn and Hoffmann in 1905 is the true cause of the disease, and that mercury, formerly given empirically, is a direct antidote by causing destruction of the parasite.

THE NAME.

This leads me to say a word or two on the three terms applied to this form of the disease: (1) congenital; (2) hereditary; (3) inherited. Of these three I greatly prefer the third. *Congenital*, besides being objectionable in form and suggestion, is not universally true. *Hereditary* suggests something that may be passed on indefinitely, of which there is no proof; whilst *inherited* implies only something derived from the parents which is detachable, like a fortune or misfortune, and this more correctly expresses the passing on of the spirochætæ to the offspring.

THE MICRO-ORGANISM.

The cause of syphilis, whether inherited or acquired, is the presence in the blood and tissues of the same organism, which can be demonstrated in the various secondary lesions, in the blood, and in the internal organs.

The *Spirochæta pallida* is a protozoon of spiral form from 4 to 20 μ in length (about half to three times the diameter of a blood-corpuscle), and $\frac{1}{4}$ μ in diameter, with a flagellum at either extremity. It is very motile, and its motility is of three kinds, a lashing, a cork-screw, and a to-and-fro movement. It stains pale pink with Giemsa's fluid, whilst the coarser highly refracting *Spirochæta refringens* with which it is often associated stains dark purple.

THE MODE OF TRANSMISSION.

The discovery of the cause of the disease necessitates the re-arrangement of our former views as to its transmission. To those who remember the revolution in thought caused by the discovery of the tubercle bacillus —how that the long-taught diathesis gradually receded into the background, and the danger of contagion by inter-communication in houses and families grew into importance—will be prepared for some change of idea in reference to syphilis.

To my mind inheritance from the father alone is now put entirely out of court, and it follows that infection of a mother by her syphilitic fœtus can never occur. For how is it possible that a spirochæta which is highly motile, and whose length averages rather more than the diameter of a blood-corpuscle, can penetrate an ovum $\frac{1}{200}$th of an inch in diameter and multiply without destroying it. I lay it down as an axiom to be demolished if you will by discussion, *that inheritance is invariably through the syphilised mother*. This is supported by Colles's law that a syphilitic infant cannot cause a chancre on the nipple of its mother when suckling.

It would seem that when virulent, the spirochætes penetrate the chorion or placenta and occasion miscarriages, macerated fœtuses, or premature births; but when the virus is attenuated by time or treatment the placenta forms a complete protection to the developing fœtus, and it is the separation of the placenta at birth which allows the infection to take place through the umbilical vein. Hence the regularity of the secondary exanthematous stage from a fortnight to three months after birth. *In these cases the separation of the placenta is the first stage, and corresponds to the chancre of acquired syphilis.*

COLLES'S LAW.

I alluded to Colles's law (which was first stated in 1837) in support of the argument that the woman was always first syphilised before the embryo or infant. The law is that a woman giving birth to a syphilitic infant cannot be inoculated with syphilis by the infant when she is suckling it—in other words, though the mother may have shown no definite signs of syphilis she is immune; whereas, the syphilitic infant put to the breast of a healthy woman may inoculate her nipple and convey syphilis to her.

Hitherto Colles's law has been used as an argument in support of the view that the mother may get a mild form of syphilis from her syphilitic fœtus, whose syphilis is supposed to be derived entirely from the father. But the law of immunity will remain equally true if it be supposed that the mother is first inoculated by the father, a large dose of the protozoon causing an obvious eruptive syphilis and a small dose a syphilis which misses the eruptive stage.

Syphilis in a man is generally admitted to be capable of transmission to a succeeding generation for a much shorter time than syphilis in a woman, and this supports the view I have stated, viz. that for transmission it is necessary that the woman be first infected.

TRANSMISSION BY MILK.

The question whether the milk of a syphilitic female may infect a healthy infant at the breast has been discussed for two and a half centuries, since Ambroise Paré, in the seventeenth century, quaintly observed: "Infants suckled by syphilitic nurses are infected by them, seeing that the milk is nothing but whitened blood, which being infected by the virus the child fed with it imbibes the same qualities." Hunter, founding his argument on ineffectual attempts to inoculate the blood of a syphilitic person, came to the conclusion that not only the blood, but every secretion derived from it, such as milk, saliva, perspiration, etc., could not convey the disease. But his experiments were inconclusive and his deductions incorrect. Pellizzari succeeded in inoculating the blood of a syphilitic person, and if Voss's experiment is to be trusted, milk has been directly inoculated also.

A remarkable case bearing on this subject I showed before the Royal Medical and Chirurgical Society in 1881, and it is published for the first time in the 'British Journal of Children's Diseases' for reference (see page 10, vol. v, No. 1). A woman, aged 30 years, gave birth to a healthy child on December the 11th, which she suckled. The following Easter her husband inoculated her with syphilis, but she continued to suckle the child. She consulted me three months later when suffering from severe secondaries, squamous eruption, sore throat, condyloma, and loss of hair, but the child showed no sign of infection. The mother was then treated with small doses of mercury. She and the child were shown when the latter was ten months old. He had continued to suckle and remained plump and in perfect health, though the mother still had patches of circinate squamous syphilide on her arms. During the two years the mother remained under my care for treatment the child showed no sign of inherited syphilis. The importance of this case rests on the fact that the mother had suckled her child for three months after her infection before any treatment was commenced, so that it cannot be argued that the infant was taking the antidote in the mother's milk with the poison, and so escaped a source of possible inoculation; but it proves that the milk of a syphilitic woman when received into the alimentary tract of an infant need not convey any infection to the child.

Transmission by Semen.

It is obvious, as the greater cannot be included in the less, that a spirochæta cannot be carried in a spermatozoon, but this does not exclude the possibility of the spirochætæ being conveyed by the fluid parts of the semen.

The early experiments of inoculation by Mireur failed, but if the recent results obtained by Finger on monkeys be correct the semen of syphilitic men is inoculable. It seems almost necessary that it should be so to account for the cases of inherited syphilis conveyed after the healing of the chancre. If the presence of the spirochætæ can be with certainty demonstrated in the semen of men suffering from recent syphilis much doubt would disappear, and, in the cases where no chancre could be traced in the woman, the probability of the infection being carried through the uterus, after the disintegration of its lining membrane at any menstrual period, would be apparent.

TRANSMISSION TO THE THIRD GENERATION.

Another question much open to discussion is whether syphilis inherited is capable of transmission to the third generation. If the tertiary symptoms, occurring ten or twenty years after inoculation, can be proved to be due to renewed activity of the spirochætæ, in certain situations, there seems to be a fair possibility of their being carried to a third generation. But the question is beset with difficulties, since you must prove the sexual purity of two persons up to the time of maturity, and these are generally persons in whom a tendency to vice is also hereditary; for it is known that a person the subject of inherited syphilis is not immune from re-inoculation after a certain period.

Hutchinson mentions eight cases that have come under his observations of persons who presented signs of inherited syphilis and who married, but whose offspring showed no evidence of the disease.

On the other hand, Edmond Fournier collected 116 cases, 59 of which he thought were to be relied upon as showing transmission to the third generation, and R. W. Taylor has published others.

Dr. D. M. Hutton brought a case before this Society which is published in the first volume of our 'Reports' and is there criticised by Dr. Ashby and others.

My own experience is limited to one case, but it is of unusual importance, as both parents showed most obvious signs of inherited syphilis which were unmistakable. A blind man attended a school for the blind, where he met a blind woman, for whom he developed a feeling of affection. They were both blind from interstitial corneo-iritis, and they both presented the typical physiognomy of the inherited disease—notched teeth and scars around the mouth. They married, and at the time the man came under my care their first child was about two months old. The blind wife was brought up to visit her husband, and seeing she was marked by the same disease, I sent for the infant, which presented no evidence whatever of syphilis, nor did it develop any symptom of inherited disease during the months I was able to keep it under observation. This case, in which there should have been double inheritance, showed, so long as I was able to watch it, complete immunity.

THE INFANT MORTALITY.

There is probably no disease responsible for such an enormous destruction of human life in its earliest stages as that caused by syphilitic parentage. But my experience shows that this mortality is greatest in those families where both parents have suffered from chancre syphilis and obvious secondaries. The severity of the infection and ineffective treatment, or lack of treatment, are the two factors which determine the mortality. I give two illustrations:

CASE 1.—E. S—, had been married eleven years, and suffered three months after marriage from rash and sore throat. The first child was stillborn twelve months after marriage, and was miscarried at the sixth month. The second child lived (the mother being at this time under treatment), but had severe snuffles and rash, is very delicate and has recurrent sores and eruptions. The third, fourth, fifth, sixth, seventh, eighth, and ninth children were all born at full time, but all died from a few minutes to within two or three months of birth. The tenth child was brought up for treatment suffering severely from snuffles, stomatitis, and coppery shiny eruption. Thus two weak children only were living out of ten.

Case 2.—R. C—, three weeks after marriage suffered from sores on vulva followed by rash and sore throat, for which she had no prolonged treatment; a pale, cachectic-looking woman. The first child, ten months after marriage, was stillborn at the seventh month. The second child was prematurely born at the eighth month and lived a day. The third child, born at full time, a fortnight later came out in large brown spots and had snuffles, and was taken to Guy's and treated by blue ointment rubbed in over the abdomen. She still lives, and at the age of eleven shows no notching of incisors or evidence of her inheritance. The fourth child did not come out in an eruption till five weeks old; he died at six weeks. The fifth child, a boy, came out in an eruption at five weeks and died at seven weeks. The sixth child was stillborn. The seventh child, prematurely born at the eighth month with an eruption upon her, survived a day. The eighth child, a girl, had a rash soon after birth and died on the eighth day. The ninth, also a girl, was born healthy, but an eruption came out at the third week. She was treated, but died at eleven weeks. The tenth child, a fine boy at birth, came out in an eruption three weeks after birth and was brought up for treatment. He was suffering from symmetrical, squamous syphilide of legs, feet, arms and ears, little on body, ulceration of mouth and buttocks, and severe snuffles. Again two living out of ten.

The Secondary Stage.

The trite definition that syphilis is a "fever diluted by time," given by the late Dr. Moxon, is applicable to the inherited as well as to the acquired disease. I argued that the primary stage is the separation of the placenta and the infection of the infant through the umbilical vein. The secondary or exanthematous stage commences from the second week to as late as the third month. It is characterised by eruptions which may vary from slight brown macular syphilide to pompholyx, by snuffles, stomatitis, condyloma, wrinkled skin and wasting, enlargement of the spleen and liver. Then follow certain changes in the bones, in severe cases perhaps epiphysitis, giving rise to pseudo-paralysis, Parrot's nodes causing natiform skull and square forehead, cranio-tabes, and bent bones.

It cannot be too strongly insisted on that the moist eruptions and ulcerations about the mouth and anus as well as the vesicular skin affections are charged with the spirochætes and highly contagious.

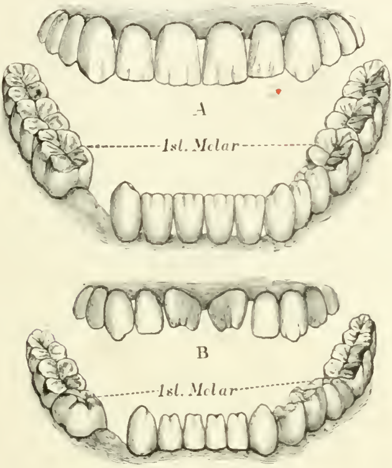
From the second to the sixth year there is commonly a rest in the symptoms that are regarded as characteristic, but the tibiæ may become thickened from periostitis, or a joint may become swollen and painful and resolve under mercurial treatment.

But now the characteristic physiognomy has been gradually formed, the flattened nose, the square forehead, the radiating lines from the mouth, the stunted figure and pallid face; and then during the second dentition we look for the three signs pointed out by our great observer Jonathan Hutchinson—the notched incisor teeth, interstitial corneitis, and syphilitic deafness. Associated with such signs or occasionally independent of them, gummatous destruction of the soft or hard plate may occur, and ulcerations of the skin and cellular tissue. Destruction of the nasal bones, caries of the forehead and skull, of the long bones and dactylitis may take place as the result of the inherited disease.

Moon's Molars.

The teeth Mr. J. Hutchinson described as so characteristic of the disease are the notched and narrowed incisors, especially the central incisors of the upper jaw. This defect is brought about by arrest in development of the central columella, of which each incisor has three. But I wish to draw special attention to the characteristic change brought about by the disease in the first molars described by the late Mr. Henry Moon many years ago,

Fig. 14.



since this change has recently been re-discovered both in America and on the Continent. The diagram which I show is taken from the fourth edition of 'Bryant's Surgery,' published in 1884, wherein the article on "Teeth" is written by Mr. Moon. He figures and describes the syphilitic first molar as "reduced in size and *dome*-shaped, through the dwarfing of the central tubercle of each cusp." He also contrasts these teeth with mercurial teeth and syphilitic-mercurial teeth. The change in the molar is of some clinical importance since occasionally it is characteristic when the incisors are normal.

EYES AND EARS.

The eyes may become affected at an early stage by a choroido-retinitis which may leave permanent changes easily recognised by ophthalmoscopic examination, and iritis, though rare, may occur during the eruptive stage. Interstitial keratitis is most frequent between the ages of six and sixteen, but may occur much later, and rarely earlier. It is highly characteristic of the inherited disease. Like the eyes, the ears are attacked by different affections in the early and late stages. During the eruptive stage otitis media may commence as an extension from the inflammation of the naso-pharynx, whilst during the second dentition a progressive deafness of labyrinthine origin may cause complete loss of hearing. Specialists in these departments will, I hope, give us some new facts relative to the syphilitic manifestations in the eyes and ears.

INHERITED SYPHILIS ATTACKING THE VISCERA.

Much work has been done in demonstrating the disease in the various viscera, not one of which appears to be exempt, and there is probably a large field still open here for further investigation. Enlarged spleen and liver associated with rickets may be proved almost invariably to be of syphilitic origin.

The disease attacks the various organs in two forms, as a small cell infiltration usually following the course of the vessels, which in its development and decline may lead to fibrosis and consequent atrophy, and as a gummatous tumour, which is less common.

When the inherited disease attacks both testicles or both ovaries at an early age, and brings about their fibrosis and atrophy, a condition known as *infantilism* is produced at a period when the sexual characteristics should be pronounced.

The physicians present will, I hope, give us much new information on inherited syphilis of the viscera.

THE NERVOUS SYSTEM.

Much difference of opinion exists as to the frequency of nervous disease dependent on inherited syphilis. Whilst some are inclined to attribute every conceivable weakness, paralysis or mental defect to this disease, others regard the nervous lesions dependent on it as infrequent. A certain proportion of epileptics, deaf-mutes and idiots, but not a large percentage, shows signs of inherited syphilis. On the other hand, syphilitic endarteritis of cerebral vessels, gummata on nerves, and sclerosis of brain and spinal cord have been observed by competent observers.

One of the most interesting observations of late years is that some cases of hydrocephalus are dependent on inherited syphilis and are curable by mercury.

THE NEED OF CRITICISM IN DIAGNOSIS.

In conclusion, I wish to insist on the importance of weighing carefully all the evidence before determining that a particular affection is due to inherited syphilis. Every deformity from dislocated hip to cleft palate, all defects such as hernia, infantile paralysis of various kinds and even nævi have been described by various writers as dependent on inherited syphilis, and as if better to cover the anomalies the term "para-syphilis" has been invented to add to the confusion.

I need scarcely waste time in combating such crude generalisations as that "all rickets takes origin in syphilis," when any puppy taken from a litter and deprived of proper food and exercise will certainly develop this disease.

Equally crude statements have been made as to other diseases. We do not deny that persons whose constitutions have been weakened by disease are liable to produce degenerates in succeeding generations; but in future the most certain test of the disease being syphilis will be the presence of the *Spirochæta pallida* in the part affected. This organism has an extraordinary persistency, producing local symptoms after lengthened periods, but happily we have in mercury and the iodides drugs which control its development and bring about its destruction.

Metchnikoff has recently shown that some hours after direct inoculation the application of a calomel ointment to the sore is sufficient to kill the organism and prevent the occurrence of secondary symptoms.

Gentlemen, I have now finished my sketch of this interesting disease in the second generation. It is the merest outline, that you may fill in the substance. I ask for new facts and new observations, for now that the cause of the disease is definitely known we have a fresh incentive to further investigation. There are many diseases still confused under a common name—just as gonorrhœa and syphilis, typhus and typhoid fever were confused generations ago—that time will unravel. But as we gain more exact, more precise and more definite knowledge of any one disease, we shall be better equipped for appreciating the symptoms and distinguishing the effects produced by others.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the* 13*th*, 1907.)

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29. "INHERITED SYPHILIS."

By GEORGE PERNET, M.D.

Although the differential diagnosis of skin diseases from syphilis in infants is very interesting, especially as regards such conditions as seborrhœic dermatitis, Jacquet's erythème érosif des nouveau-nés, etc., and late manifestations of congenital syphilis about the skin chiefly, I do not propose to touch on these points, especially as I have dealt with them in a monograph on the subject (1). The chief interest of the present discussion is, I consider, heredity, the most important feature of syphilis now that the causal organism has been discovered. I have a strong objection to the term "hereditary syphilis," which is constantly used on the Continent, the English term "congenital syphilis," though not scientifically correct, being preferable. I think there is much vague talk about heredity, a subject about which very little is really known. A work such as that by Yves Delage shows how complex is the study of heredity. Then, again, there are Beard's views as to the antithetic alternation of generations in Metazoa and the wonderful germ-cells (see Beard's "Morphological Continuity of Germ-cells as the Basis of Heredity and Variation," 'Rev. of Neurol. and Psychiatry,' vol. ii, 1904, etc.). By "heredity" in the present connection I mean the conveyance of syphilis by the spermatozoon directly to the ovum. I contend that this does not take place. My view, which is supported by many facts, is that syphilis transmitted to the child is *always of maternal origin*, and that it is not a direct paternal infection. The mother of the child must be infected with syphilis before the child can receive it from her, and thus I am in agreement with what Mr. Lucas * stated on this important point. I am particularly anxious that the subject should be discussed before this Society. In congenital syphilitics the liver is full of spirochætæ carried from the mother by the umbilical vein. I desire to refer to some cases of interest. An instructive case has been recorded by Jullien of a syphilitic man with supernumerary fingers whose infected wife was delivered of a syphilitic child, the child having supernumerary fingers also. But the same man's mistress had borne a healthy child, also with supernumerary fingers (2). I consider there is no doubt about the paternity in these two cases. Further, as to paternity there is always the possibility of a fallacy in arriving at a conclusion. The saying that it is a wise child that knows its own father has much truth in it. I believe that Colles's law is a law. The supposed exceptions when critically reviewed will not hold water. It is well known that a man with florid syphilis may, if he has no lesion about the genitalia, beget a healthy child. A very interesting instance of this kind is recorded by Matzenauer, who has written a most exhaustive account of congenital syphilis, which all should read (3). A healthy child was born, and the wife was not infected, although, as I say, the man was in a florid state of syphilis. He did not communicate syphilis to his wife, but at two years of age this child became accidentally infected from a sore on the lip of its own father. In that kind of case the wife might, of course, become infected by the husband subsequently (4). With regard to the sperm, Mr. Lucas * mentioned Finger's experiments, but Erich Hoffmann has not confirmed them. It is unfortunate that Finger did not examine the sperm for the treponema. But though the sperm may contain the parasites, I should not call infection by sperm heredity; I take it heredity means infection of the human cell ovum by the human cell spermatozoon. On the question of heredity I wrote a letter not long ago to the 'British Medical Journal' (5) in reference to an address on Mendelian heredity by Mr. Bateson (6), in which the latter referred to "pébrine," saying it was possible that some of those diseases might be due to the transmission of the disease germ through the reproductive cells as in "pébrine." That is the kind of statement brought up by those who wish to prove that syphilis is hereditary. Neither I nor Matzenauer have been able to verify the point, and when I called Mr. Bateson's attention to this the latter said: "Mr. Pernet properly insists that transmission of a disease germ in the egg is not the same thing as in the reproductive cells." That is very important. Of course, the egg may be infected afterwards, but that is not heredity in the sense defined. Mr. Bateson admitted he was

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* 'British Journal of Children's Diseases,' vol. v, p. 3.

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* *Loc. cit.*, p. 5.

wrong in using the more inclusive expression. There is no truth in the statement constantly being made that syphilis is on all fours with "pébrine." Heredity in tuberculosis (7) and leprosy have been given up, and so must it be in syphilis. In this connection I would refer to Gärtner's observations on the number of spermatozoa in sperm. Gärtner came to the conclusion that on the average only 1 out of about 85,000,000,000 spermatozoa had the chance of fertilising an ovum (8). I believe Colles's law to be accurate, and that great credit is due to the enunciator of it. Everyone interested in the subject of syphilis should read Colles's original remarks. Colles himself came across a case which appeared to contradict his conclusions, but further investigation showed that it was not an exception to what he considered the course of events in such cases. I have looked up several cases which were said to be exceptions. In one case I found the disease was not syphilis, but pemphigus neonatorum; the mother was infected with syphilis afterwards. Among others there is a most instructive case of Erich Hoffmann's, which should be consulted in this connection (9). I hope the Society will come to a clear understanding of the subject, particularly from the point of view of heredity, which I regard as the central theme in syphilis in the new-born. I repeat that in my opinion congenital syphilis in the child is derived from the mother, and that Colles's law is really a law.

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(*Read at the special meeting for the discussion of "Inherited Syphilis," December the 13th,* 1907.)

30. INTERSTITIAL KERATITIS FROM A MODERN STANDPOINT.

By SYDNEY STEPHENSON, C.M.

It would be difficult or impossible to over-estimate the diagnostic importance which attaches to some of the ocular stigmata of inherited syphilis. By their discovery the nature of many and many an obscure case has been cleared up. I can recall a couple of cases of necrosis, recognised as syphilitic in one instance by the finding of disseminated choroido-retinitis, and in the other by the advent of interstitial keratitis. The most striking case with which I am acquainted, however, was related by Edmond Fournier in his monograph on late hereditosyphilis ('Recherché et Diagnostic de l'hérédo-syphilis tardive,' 1907, p. 325). A man, aged 34 years, was believed to be suffering from sarcoma of the pelvis, declared by three competent hospital surgeons to be beyond the reach of operation. The patient's elder brother, aged 36 years, was examined by my friend, Dr. Antonelli, with the ophthalmoscope, and found to be affected with positive stigmata of congenital syphilis. The result of this discovery was happy in the extreme. For after two months' treatment by mercurial injections and potassium iodide, the pelvic growth disappeared without leaving a trace and the patient was restored to perfect health. His remark was amply justified when he exclaimed : " J'ai été sauvé par les yeux de mon frère."

Of all the ocular manifestations of inherited syphilis, none is better known or easier to recognise than diffuse interstitial or parenchymatous keratitis.

The disease is not exactly a common one. For example, Greeff (' Die Keratitis interstitialis in ihren Beziehungen zu Allgemeinerkrankungen,' 1897) collected figures respecting 36,385 eye patients, and found that interstitial keratitis had been diagnosed in 279—*i. e.* in 0·77 per cent. of the total number. Again, among 5142 eye patients seen by me at the Queen's Hospital for Children, 49, or 0·95 per cent., were affected. The proportion would naturally be somewhat higher at a children's hospital than elsewhere.

The disease has been familiar to surgeons for many years. William Mackenzie (' A Practical Treatise on the Diseases of the Eye,' 1830, p. 419), who takes in ophthalmic science the position occupied by Sir Thomas Watson in general medicine, was acquainted with the malady, which was called by him "scrofulous corneitis." Mackenzie noted its coincidence with deafness, a peculiar hoarseness of voice, swollen lymphatic glands, nodes on the tibiæ, and effusion into the bursa beneath the tendon of the extensores cruris, symptoms some of which we now recognise to be manifestations of inherited syphilis.

It was, however, reserved for Jonathan Hutchinson (' A Clinical Memoir on Certain Diseases of the Eye and Ear, consequent on Inherited Syphilis,' 1863) to show that interstitial keratitis was " almost always a direct result of inherited syphilis," and to give a description of its clinical characters and morbid associations that is as faithful today as when he wrote it forty-four years ago. In particular, Hutchinson drew attention for the first time to the diagnostic value of certain malformations of the teeth often met with in cases of interstitial keratitis.

A few years later James Dixon (' A Guide to the Practical Study of Diseases of the Eye,' third edition, 1866, p. 95), went farther, and proposed to substitute for Hutchinson's name, "chronic interstitial keratitis," that of "syphilitic keratitis," since there existed, he said, "no special form of keratitis connected with acquired syphilis."

Still more recently Hutchinson ('Syphilis,' London, 1889, p. 75) has claimed that "interstitial keratitis, in its typical form, is always a consequence of syphilis, and is in itself sufficient for the diagnosis."

To some extent, however, the pendulum of professional opinion has now swung back to the views expressed so many years ago by William Mackenzie. The view current to-day, especially upon the Continent, is that while parenchymatous keratitis is usually a manifestation of inherited syphilis, yet a proportion of the cases are due to other factors, among the more important of which are tuberculosis, acquired syphilis, influenza, malaria and trypanosomiasis. In a word, it will scarcely be denied that interstitial keratitis is the local manifestation of some general disorder, be it syphilis or otherwise.

It will clear the ground if a few figures be quoted as to the relative frequency of inherited syphilis as a cause of interstitial keratitis. Davidson found 20 per cent., Alexander 35·3 per cent., Fournier 41·5 per cent., Hirschberg 61 per cent., Saemisch 62 per cent., Horner 64 per cent., Mauthner 80 per cent., Bosse 81 per cent., and Silex 83 per cent. of the cases to be due to a specific cause in the shape of inherited syphilis. Pfister ('Klin. Monatsbl. f. Augenheilkunde,' xxviii, 1890, p. 114) found certain evidence of hereditary lues in 40·8 per cent. of his 130 cases and presumptive evidence in 23·8 per cent.—total 64·6 per cent. Nettleship ('Diseases of the Eye,' fifth edition, 1890, p. 120) found personal (54 per cent.) or family (14 per cent.) evidence of inherited syphilis in 68 per cent. of an unspecified number of cases, and in most of the remaining 32 per cent. there were strong reasons for suspecting its existence. My own figures dealing with 101 cases gave inherited syphilis in 70, or 69·3 per cent.

Other statistics might readily be quoted, but enough have been given to show that, broadly speaking, adequate evidence of inherited syphilis may be obtained in about two-thirds of all cases of parenchymatous keratitis.

With regard to the frequency of tuberculous interstitial keratitis, some interesting figures have recently been published by H. Rabiger (ref. in 'Archives of Ophthalmology,' November, 1907, p. 875). In 349 cases of keratitis observed at the University Polyclinic at Berlin, tuberculosis was found with certainty in 11 per cent. and with probability in 9·7 per cent.—total, 20·7 per cent. In my own series of 101 cases tuberculosis was identified as the cause of the keratitis in 10—that is, in 9·90 per cent. The more extended employment of Calmette's serum test will doubtless help us to recognise such cases with more certainty.

It would appear that interstitial keratitis is by no means rare as a late, tertiary consequence of acquired syphilis. The connection was first mentioned by Velpeau in 1840 ('Maladie des Yeux'), and was described in 1861 by Follin ('Pathologie Externe,' t. 1, p. 708, 1861)—that is to say, two years before the publication of Hutchinson's famous memoir, in which he commented on "the entire absence of interstitial keratitis from the *rôle* of tertiary symptoms of acquired syphilis" (*loc. cit.*, p. 221). In a later work ('Syphilis,' 1889, p. 237), however, Hutchinson modified this view and recognised the possibility of an acquired interstitial keratitis.

According to figures collected by Pfister from Professor Haab's klinik (*loc. cit.*), of 130 cases of keratitis 3·8 per cent. (2·3 per cent. certain and 1·5 per cent. somewhat doubtful) were due to the acquired disorder, while Ancke ('Centralbl. f. Prak. Augenheilkunde, 1885, p. 360) found the proportion in 100 cases to be as high as 10 per cent. Among my series of 101 cases of parenchymatous keratitis, 4, or 3·96 per cent., were unquestionably due to acquired syphilis.

It is important to note that such cases have been met with even in children. For example, Mauthner ('Zeissl's Lehrbuch der Syphilis,' p. 279) saw a case of interstitial keratitis in a suckling that had contracted syphilis from its nurse. Moreover, Trousseau ('Annales d'Oculistique,' September, 1895, p. 206) has mentioned the case of a boy, aged 8 years, who was infected by his nurse with syphilis. The result was a very severe attack of bilateral keratitis. Lastly, I reported a case of the kind ('The Ophthalmoscope,' vol. i, 1903, p. 169) in a girl, aged 12 years, who had suffered from a chancre on one upper eyelid two or three months after birth.

It may be noted, as an interesting point, that quite a number of cases of interstitial keratitis have been known to follow an indurated chancre on the eyelid or conjunctiva.

The name "interstitial keratitis" is good in so far as it directs attention to a prominent feature of the malady—namely, to opacities "like microscopic masses of fog," situated at various levels in the substance of the cornea. It suggests, nevertheless, a mistaken view as to the pathology of the condition (Fuchs, v. Hipple, Pflüger, de Lapersonne, C. D. Marshall, Parsons, etc.). The available evidence indicates that the ailment is primarily an inflammation of the tissues of the ciliary body, which forms (as everybody knows) the middle part of the uveal tract, of which the other parts are the iris and the choroid. From the ciliary body inflammation spreads forwards to the iris as well as to the deeper layers of the cornea, and backwards to the anterior parts of the choroid. Keratitis and the commonly associated choroiditis and iritis therefore are, strictly speaking, conditions secondary to an inflammation of the ciliary body. The underlying process may conveniently and accurately be described by the expression "anterior uveitis."

If this view be not grasped, it becomes difficult to understand some clinical features of the disease. For example, in my experience it is not very uncommon for the ailment to begin with what appears to be an accumulation of pus in the anterior chamber of the inflamed eye. This hypopyon, of course, represents an exudation from the inflamed ciliary body, which has passed through the pupil and has thereby gained the anterior chamber. Another early sign is the existence of deposits on Descemet's membrane, the "aquo-capsulitis" of the older and the "keratitis punctata" of the more modern writers, and this, again, is to be regarded as the expression of an exudation from the ciliary body. The occasional bleeding into the anterior chamber (hyphæma), which may usher in an attack of parenchymatous keratitis, is almost certainly due to the rupture of some small distended vessel in the ciliary body. None of these appearances can be explained on the theory that the disease is primarily one of the cornea.

Further, the iritis which often precedes or accompanies the keratitis can be best accounted for by an extension forwards from an inflamed ciliary body.

A similar remark applies equally to the choroiditis which is frequently found when the cornea has cleared enough to allow of an examination of the fundus with the ophthalmoscope. In cases where choroiditis is known to precede by months or years the development of keratitis, the backward extension to the choroid has antedated the forward extension to the cornea, or has possibly occurred independently of the ciliary inflammation. Cases of this type are sometimes of an unusually severe type. The following is an example: John O—, aged 8 years, first seen on January the 5th, 1893. A typically syphilitic subject with "bossy" forehead, rhagades, and dwarfed and notched upper central incisors, and a tendency to ptosis. Sight equalled about one-fifth of the normal, as the result of widely-spread bilateral chorido-retinitis. In September, 1893, the right eye developed interstitial keratitis, and seven months later (April the 25th, 1894) the left eye followed suit. On May the 30th each knee-joint became distended with fluid. The corneæ presented a peculiar appearance, since an irregular area towards the centre, 5 mm. at its widest part, resembled damp wash-leather, and was everywhere surrounded by a zone of plum-coloured vascular tissue—a confluent "salmon patch," in short. Viewed from the side, the cornea projected forward cone-wise, the blunt apex of the cone being formed by opaque cornea and its sides by the vascular patch. By this time the lad had become somewhat deaf, without discharge from the ears. On June the 19th a portion of the grey opaque central area had exfoliated. Deafness was increasing. Both ankles had become swollen. In August, 1894, the lad developed delusions, which became very pronounced towards the end of that month. He used vile language, insisted upon getting out of bed for the express purpose of breaking the windows of the ward, or for that matter anything else he could grope his way to, for by this time he was to all intents and purposes blind. John O— was then transferred from the Ophthalmic School to another institution, where "he developed most acute mania, and had delusions that people were coming to kill him, and that there were bloodhounds in the cupboard." In 1902—that is, nine years after I first saw him—the patient was an inmate of Darenth Asylum, quite blind, and suffering from imbecility with occasional attacks of excitement. One of his eyes had been removed owing to an injury.

The case just described presented several peculiarities, and amongst them ulceration of one cornea. This complication, in my experience, is not common. For instance, among my ninety-seven cases* of interstitial keratitis, it occurred in four—that is to say, in 4·12 per cent. That it might occur was known to Hutchinson, who alluded to one or two cases on page 29 of his oft-quoted memoir. W. Spencer Watson ('Ophthalmic Hospital Reports,' vol. iv, p. 296), writing a few years after Hutchinson, reported an instance of unilateral ulceration observed in a series of twenty-five cases of interstitial keratitis. R. Ancke ('Centralbl. f. Prak. Augenheilkunde,' 1885, p. 360) had one example of ulceration among 100 cases of interstitial keratitis. E. T. Collins ('Ophthalmic Hospital Reports,' vol. xi, 1887) described four cases in corneæ which showed extreme vascularity, and in which a small central island of non-vascular tissue was alone left. This islet became yellow and then ulcerated. In one of Collins' cases there was perforation, but cicatrisation took place in the other three. His patients showed unmistakable signs of inherited syphilis, either in the teeth or the physiognomy.

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* Four cases due to acquired syphilis omitted.

In a majority of cases of parenchymatous keratitis one cornea is first attacked and the other cornea, according to Hutchinson (*loc. cit.*, p. 29), "after from one to two months." But it should be borne in mind that the interval may be, and often is, much longer than this. The extreme instance reported by Consiglio ('Beitrage z. Augenheilkunde,' May, 1905), where the interval amounted to twenty-six years, must be almost unique, although in his case the evidence of inherited syphilis was not altogether conclusive. My own series includes one case where the interval amounted to about seven years. The facts follow: Edmund D—, aged 13 years, seen on December the 22nd, 1899, with early interstitial keratitis affecting the right eye, which had been divergent since infancy After making a nearly complete recovery, the right eye relapsed in September, 1905. On November the 30th, 1906, the following note was made: "The left eye has been affected for the last fortnight, *i.e.*, there has been an interval of seven years between the attacks in the two eyes. The patient is deaf, and his teeth are typical of inherited syphilis. Hutchinson's triad is, therefore, present."

All observers agree that parenchymatous keratitis is essentially a disease of childhood. On this point there is no difference of opinion. It is, nevertheless, relatively rare in very young children, and equally so after mature age has been reached. Hutchinson, for example, had never witnessed its occurrence earlier than two years or later than twenty-six years. It is most frequent between the ages of five and eighteen years (Hutchinson) or of six and fifteen years (Nettleship).

The age-incidence of my ninety-seven cases, arranged in quinquennial periods, comes out as follows:

Period.	Number.	Percentage.
1 to 5 years	6	6·19
5 ,, 10 ,,	38	39·18
10 ,, 15 ,,	26	26·80
15 ,, 20 ,,	14	14·43
20 ,, 25 ,,	8	8·25
25 ,, 30 ,,	2	2·06
Over 30 years	3	3·09

An inspection of the foregoing figures will at once show that of my ninety-seven cases four fifths, or 80·41 per cent., occurred between the ages of five and fifteen years. My series includes one case in a woman, aged 36 years, whose syphilitic inheritance was attested by notched upper central incisors, rhagades at angles of the mouth, and by a node on the frontal bone, 1 inch above the inner end of the left eyebrow. R. Marcus Gunn ('The Polyclinic,' December, 1902), reported a case in a patient aged 36 years; Pfister (*loc. cit.*) in a man, aged 37½ years; and Greef (*loc. cit.*, p. 13) in a man, aged 38 years.

The rule, that syphilitic parenchymatous keratitis is relatively commoner in females than in males, is supported by an analysis of my own cases, 97 in number. Of the total, 37 (38·14 per cent.) were in males and 60 (61·86 per cent) in females. It is perhaps worth noting that the same curious disproportion between males and females applies also to juvenile tabes dorsalis. Thus, Cantonnet ('Archives d'Ophtalmologie,' November, 1907), as the result of an analysis of 88 cases found that females were affected in 63·6 per cent, and males in 37·3 per cent.

It should be noted that the figures given by Baker and Story ('Ophthalmic Review,' November, 1885), and by Ancke ('Centralbl. f. Prak. Augenheilkunde,' 1885,

p. 360), respectively, form an exception to the general experience as to the age-incidence of keratitis, as stated above.

The discovery of the specific cause of syphilis in the shape of the *Treponema pallidum*, together with the possibility of inoculating apes, rabbits,* and dogs with the virus, has widened our conception of the morbid processes that lead up to the development of interstitial keratitis. The disease in question has been produced experimentally in certain animals by the implantation of syphilitic products. Indeed, prior to the discovery of the treponema, P. Salmon (ref. in 'Archives d'Ophtalmologie,' April, 1905, p. 1263) observed iritis and keratitis thirty-three days after a monkey had been inoculated with a syphilitic papule from man.

We know that the tissues and organs of the syphilitic fœtus or baby are literally flooded with the treponema, large numbers of which have been found in the placenta, the umbilical cord, the blood of the umbilical vein, and, especially the liver. The organisms that escape the liver are distributed by the fœtal circulation to every part of the body, where they determine this or that syphilitic lesion. The treponema has been found in cutaneous lesions, as pemphigus, by Levaditi and others; in peri-oral ulcerations by Elizaldi and Wernicke; in glandular lesions by Panea and Babes. That is by no means all, for apart from definite lesions such as those named, the treponema had been found in every internal organ so far examined with the microscope. As regards the eye, the findings from our present point of view are highly suggestive. Thus, Hans Bab ('Deutsche Med. Wochenschrift,' November the 29th, 1906) examined the eyes of three specific still-born babies. Preparations were treated by the silver impregnation method, and large numbers of the spirochætes were found in all the tissues of the eye, with the exception of the vitreous humour and the crystalline lens. It is important to note that the corneæ contained a large number of the organisms. Again, H. Schlimpert (*ibid.*, 1906, p. 1942) succeeded in discovering the treponema in various parts of the eyes of syphilitic fœtuses. Lastly, spirochætes have been demonstrated in the tissues of seemingly unaffected eyes of syphilitic fœtuses and babies by some other observers, including Peters, Gierke and Stock, and myself.

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* As long ago as 1881 Paul Haensell (v. Graefe's 'Archiv f. Ophthalmologie,' vol. xxvii, III, p. 93), showed that syphilitic virus could be successfully inoculated into the iris and cornea of the rabbit.

It would thus seem probable that if the child survive, these micro-organisms (possibly in some different morphological form) lie dormant in the tissues of the eye, more especially in the ciliary body and the cornea. They cause no mischief until some exciting cause, of a local or general nature, lowers the resistance of the part, and allows the treponema to come into action. The result is an attack of interstitial keratitis.

In this view we can explain an observation made by many surgeons, namely, that a slight injury to the eye may precipitate an attack of parenchymatous keratitis. I well remember the first case of this kind that happened in my practice. A girl, aged 11 years, was admitted into the Ophthalmic School, Hanwell, for trachoma, pannus, and lead opacities of each cornea, in consequence of which the child was almost blind. Her syphilitic diathesis was attested by (*a*) notched central incisors of the upper jaw; (*b*) scars radiating from the mouth, the so-called "Fournier's cicatrices"; (*c*) "epithelial denudations"* about the skin of the upper lip and nose; (*d*) slightly enlarged lymphatic glands; and (*e*) cicatrices about the anus and buttocks, the so-called "Parrot's cicatrices." On March the 16th, 1897, under cocaine, the metallic incrustations were more or less scraped away from the right cornea by means of a small, sharp scoop. The little operation, somewhat to my surprise, was followed by redness of the eye, and by increased haziness and vascularity of the cornea—in a word, by the clinical appearances of an ordinary parenchymatous keratitis. On May the 4th—that is, forty-nine days after the operation—the condition was typical of interstitial keratitis. In view of ætiology, the case was an interesting one, and so on June the 16th the operation was repeated on the other eye. I am bound to add that the second intervention was not followed by interstitial inflammation. Be that as it may, the association between slight injury to the eye on the one hand, and parenchymatous inflammation on the other, is far from uncommon, as will be obvious from the fact that I possess notes of fifteen such cases.

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* The expression "epithelial denudation" I apply to those superficial cicatrices, whose favourite site is at the junction of the ala nasi with the upper lip. Their significance and diagnostic value is the same as Fournier's cicatrices.

Interstitial keratitis may supervene during the course of illnesses, such as influenza and typhoid fever. Here are one or two cases in point:

Ellen W—, aged 11 years, was admitted into the Queen's Hospital for Children, under my colleague, Dr. James Taylor, on account of typhoid fever. On the seventeenth day of the fever (approximately) the left pupil was noticed to be contracted and the cornea of the left eye rather dull. When seen by me five days later there could be no doubt that the case was one of parenchymatous keratitis. The girl had suffered from periostitis of the right tibia for several weeks. Three months after the first eye had been attacked the other cornea went through a much milder attack of keratitis.

Florence G—, aged 8 years, a child who presented rhagades at the angles of the mouth, a *café au lait* complexion, and suspicious incisor and molar teeth, in addition to slight deafness, was brought to the Evelina Hospital on March the 15th, 1905, for a relapsed interstitial keratitis of her right eye. The history was to the effect that three years before, about a fortnight after her discharge from an Asylum Board Hospital, where she had spent three months on occount of enteric fever, both eyes became inflamed, one soon after the other. The condition had slowly improved until a relapse a few weeks before the child was brought to me.

I have also seen interstitial keratitis follow a whitlow so closely as to suggest a connection between the two affections.

It is now, I think, generally recognised that interstitial keratitis may relapse—or, more correctly, recur—in one and the same patient. Manasse in 50 cases had 8 recurrences, or 16 per cent., and v. Hippel among 87 cases 15 recurrences, or 17·25 per cent. The proportion in my own cases, many of which remained under observation for long periods of time, was somewhat higher, namely, 22 per cent. As to the explanation of recurrences, we must assume that spirochætes have remained dormant in the tissues of the cornea as a sequel of the primary attack. I believe that recurrences are commoner in cases originally treated without than with mercury, and I am confident that, as a rule, they are more difficult to manage than the original attack.

Experimental keratitis.—Bertarelli ('Presse Médicale,' August the 22nd, 1906) obtained a positive result by inoculating the cornea of rabbits with an emulsion of human chancres. The animal's corneæ showed numerous spirochætes, and, in addition, a pronounced leucocytic infiltration, which extended into the seemingly unaffected parts of the cornea. Scherber and v. Benedek ('Münch. Med. Wochenschrift,' June the 14th, 1906) produced nodular iritis and interstitial keratitis by inoculating the anterior chamber of rabbits with syphilitic virus. Most suggestive experiments were reported during the course of 1906 by Greeff and Clausen ('Bericht der Oph. Gesellschaft,' Heidelberg, 1906), who inoculated the eyes of apes and rabbits with syphilitic material, and after the lapse of some weeks observed the development of a kind of parenchymatous keratitis. In the earlier cases numerous spirochætes could be demonstrated, but the organism could not be found in the more advanced cases. The authors concluded that the corneal opacity was the outcome of a leucocytic invasion, which attacked and eventually destroyed the micro-parasite.

In a second communication ('Deutsche Med. Wochenschrift.,' 1906, Nr. 36) Greeff and Clausen concluded that the pathogenic agents of syphilis multiplied in the cornea, and thus caused an invasion by leucocytes, which finally exterminated the micro-organisms. Lastly, Hoffman and Bruening ('Deutsche Med. Wochenschrift.,' April the 4th, 1907) produced keratitis by the inoculation of a rabbit's eye with a morsel of a human chancre, and obtained a somewhat similar result when an emulsified chancre was introduced into the anterior chamber. Smears from the diseased cornea, when stained by Giemsa's method, showed the *Spirochæta pallida*, thereby proving the essentially syphilitic nature of the keratitis.

Panas ('Archives d'Ophtalmologie,' 1871, p. 577) taught that interstitial keratitis was a dystrophic symptom, the indirect cause of which was to be sought not only in ancestral syphilis but also in lymphatism, scrofula, gout and arthritism. It is now suggested, mainly as the result of experimental and histological researches, that it is, in reality, an infection of the tissues of the cornea with this or that specific organism, the most important of such being the *Treponema pallidum*, the tubercle bacillus, the *B. influenzæ*, the *Plasmodium malariæ*, and the parasite of trypanosomiasis.

As regards trypanosomiasis, in dogs and goats suffering from nagara and dourine, closely allied to, if not actually identical with, sleeping sickness, interstitial keratitis is the commonest corneal affection. The changes have been shown by Morex ('Annales d'Oculistique,' December, 1906) to be due in these animals to the growth of the parasite in the intermitted spaces of the cornea.

In brief, it can nowadays scarcely be doubted that syphilitic interstitial keratitis is due to the presence in the parenchyma of the cornea of the causal agent of syphilis. The treponema, however, has yet to be demonstrated in that structure. The hiatus on this vital point is not to be wondered at, since eyes are seldom if ever removed during the height of parenchymatous keratitis, nor do patients usually die during the course of that affection. Speaking for myself, I have failed to find the spirochæta in scrapings from eyes affected with keratitis; but then superficial parts only were removed. Despite the known difficulty of recognising the treponema in tertiary syphilitic lesions, it is safe to predict that sooner or later the organism will be discovered in the tissues of the cornea. The chain of evidence, which still lacks one link, will then be complete.

Aphorisms respecting Interstitial Keratitis.

(1) Interstitial keratitis does not form more than 1 per cent. of the cases met with in a special department for diseases of the eye.

(2) The disease can be shown in about two thirds of all cases to be associated with signs of inherited syphilis, of which the commonest are the dental, facial, and ocular stigmata. Other important causes are tuberculosis, acquired syphilis, influenza, malaria, and sleeping sickness.

(3) Given a predisposing cause, the affection may be excited by practically anything that lowers resistance, either local or general.

(4) Interstitial keratitis is not a primary affection of the cornea, but is probably in every instance secondary to changes in the anterior part of the uveal tract.

(5) In three fourths of all cases the ailment is bilateral. In cases due to inherited syphilis bilaterality is the rule. The interval between the two eyes being attacked, however, may extend to several years.

(6) The disease is nearly twice as frequent in females as in males.

(7) Four fifths of the cases occur in children between the ages of 5 and 15 years. The greatest incidence of

the disease is observed between the ages of 5 and 10 years.

(8) Ulceration of the cornea occurs in a notable percentage of all cases.

(9) Recurrences occur in perhaps one fifth of the patients, are commoner in cases treated without than with mercury, and are often very difficult to manage.

(10) Interstitial keratitis is due to the lodgment and multiplication in the cornea of the *Treponema pallidum*, derived in the first instance from the uveal tract.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the 13th*, 1907.)

31. "INHERITED SYPHILIS."

By H. G. ADAMSON, M.D.

(INTRODUCED.)

THE importance of the careful study of the skin eruptions which occur in the napkin region of infants is not sufficiently appreciated. As a consequence of want of knowledge of these eruptions it is a common error to mistake many forms of non-specific eruption for those of congenital syphilis. The seriousness of such mistakes is obvious—not only is the infant put under a long and unnecessary course of mercurial treatment, but it is a grave matter that the parents should from henceforth be regarded as syphilitics. Non-familiarity with these eruptions may also lead to the mistaking of a syphilitic eruption for a non-specific eruption, with the result that the infant is not properly treated at an early stage, and later suffers from the more harmful effects of the specific virus. But errors in this direction are less common, for the tendency is rather to regard any sort of eruption about the buttocks and genitals of an infant as being probably of syphilitic nature.

If we turn to the text-books we get really very little definite information in this matter of the diagnosis of congenital syphilitic eruptions. We are told to carefully distinguish them from intertrigos and from eczemas, and as a means of distinction we are generally given the very misleading piece of advice that, while the non-specific eruptions are limited to the flexures and to parts covered by the napkin, syphilitic eruptions occur also upon parts beyond the napkin region. Now, as a matter of fact eczema does not occur upon the buttocks of an infant—its characteristic seat is the face and scalp; while intertrigo is a loosely used term which may cover any sort of abrasion or moist lesion in the flexures, either non-specific or syphilitic.

There are, however, several distinct and characteristic forms of eruption of common occurrence which may be, and which often are, confused with those of congenital syphilis; and it is essential not only to distinguish these eruptions from the syphilitic eruptions, but also, for their proper treatment, to recognise them individually.

In one group of these eruptions the lesions are due probably to local irritation, combined with a gastro-intestinal toxæmia. In others they owe their origin to local microbic infection. Broadly, they may be divided into the following groups:

(1) Simple infantile erythemas (*Dermites simples infantiles* of Jacquet).

(2) Streptococcic impetigos.

(3) Seborrhœic dermatitis.

SURFACE ANATOMY OF THE NAPKIN REGION.

Before proceeding to the study of these eruptions it will be of advantage to take note of certain features in the superficial anatomy of these regions.

(1) The *napkin-region* in an infant may be said to include not only the *lower abdomen, the buttocks, the genitals,* and the *thighs,* but also other parts which come

into contact with the napkin from outside when the infant is in the flexed position, namely, the *calves* and the *heels*.

(2) The area covered by the napkin comprises a number of prominent convex surfaces, separated by more or less deep sulci. These prominent convex surfaces are: In front: (*a*) the lower part of the abdomen; (*b*) the genitals and perinæum (both parts more prominent in an infant than in an adult, and, moreover, subjected to pressure and friction from the fold of napkin which is brought forward between the thighs); (*c*) and (*d*) the upper and lower halves of the thigh, separated by a deep fissure which is present only in babies. The prominent convex surface behind are (*a*) the buttocks, (*b*) the scrotum in the male, (*c*) the thighs, (*d*) the calves, and (*e*) the heels.

(3) The convex surfaces are subjected to friction and pressure from the napkin; the flexures or sulci between them are protected from friction, but afford warm, moist areas for the growth of micro-organisms.

I. SIMPLE ERYTHEMAS OF THE NAPKIN REGION (DERMITES SIMPLES INFANTILES OF JACQUET).

There are certain eruptions of common occurrence about the buttocks, genitals, and thighs of infants which have been studied and described by many of the great clinical masters of the past, including Trousseau, Henoch, and Parrot, but which have only within more recent years been thoroughly defined and clearly differentiated from the eruptions of congenital syphilis by the work of Jacquet in Paris and by other French observers stimulated by his writings.*

These eruptions consist primarily and essentially in an erythema occupying the prominent convex surfaces of the napkin region, together with modified forms, erosions, pseudo papules, and ulcerations, the result of secondary changes.

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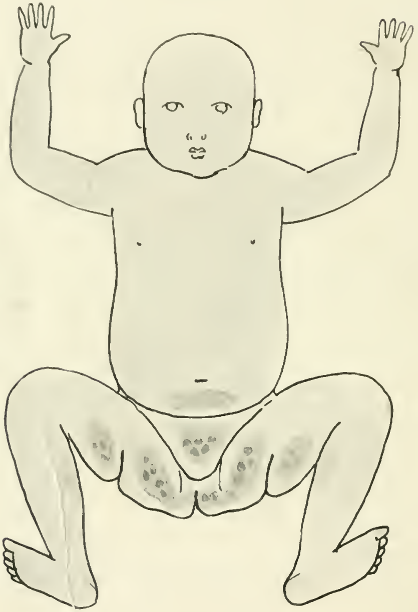
* Lucien Jacquet, 'Traité des Maladies de l'Enfance,' Graucher et Comby, t. iv, p. 714, 1905.

Jacquet has grouped these eruptions as follows:—

(*a*) Simple erythemas.
(*b*) Erythemato-vesicular or erosive.
(*c*) Papular or post-erosive.
(*d*) Ulcerative.

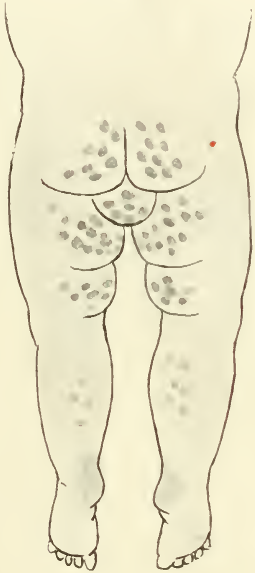
FIG. 15.



It will be more convenient to describe these forms separately, although it must be understood that they are actually stages of one process and that they may overlap or co-exist in the same patient.

(*a*) *Simple erythemas.*—These are characterised by an erythema occupying the prominent convex surfaces of the buttocks, of the thighs, of the scrotum or vulva, and extending sometimes on to the lumbar region, the lower abdomen, and the calves and heels. In some cases even the back and shoulders, where they form points of pressure as the child lies on its back, may be affected. In very mild cases only the genitals, the inner sides of the thighs, and the perinæum, may be the seats of the erythematous patches.

Fig. 16.



The erythema forms usually an uniformly shining red surface upon the convex areas, leaving free in marked contrast the flexures and sulci between. Sometimes there may be here and there excoriations, and some scaling or crusting in parts.

This type is seen mostly in very young infants (Fig. 15).

(*b*) *Erythemato-vesicular or erosive forms.*—In these there is a basis of erythema as in the first type, but in addition, towards the centre of the convex areas there are small bright red disc-like erosions in groups of two or three up to a dozen or more. The erosions may coalesce and so form polycyclical areas. Generally on close examination of the margins of the erythematous areas there may be found small, flat, more or less perfect vesicles, which represent apparently the early stage of the erosion.

This type of eruption was minutely and accurately described by Parrot. It and the succeeding type are commonly seen in babies of a few months old (Fig. 16).

(*c*) *Erythemato-papular or post-erosive.*—This form was also carefully described by Parrot, who, however, wrongly regarded it as a syphilide (lenticular syphilide).

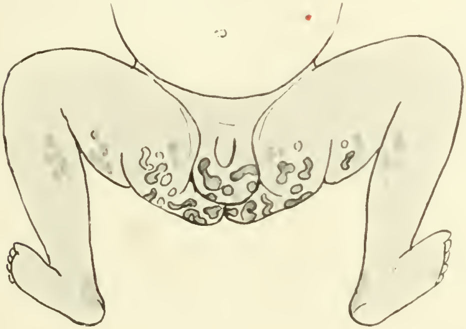
Jacquet has demonstrated that it is really a later stage of the last form in which the erosions have thrown up flattened granulations, giving the lesions the appearance of papules.

This eruption occurs like the preceding type over the prominent parts of the convex surfaces. The lesions consist of flat, bright red or dull red papules (or pseudopapules) arranged in groups, which may or may not be set upon an erythematous base. These groups of pseudopapules are seen upon the buttocks, the upper half of the thigh, the lower half, the calf, and on the genitals. They are never found in the flexures or sulci dividing the convex areas. They may be associated with erythema of the heels and of the lower abdomen (Fig. 16).

(*d*) *Ulcerating forms.*—These are less common than the preceding, although not altogether rare in hospital practice. In these cases the erosions, instead of granulating, become still further advanced, so that ulcers are formed. The ulcers are punched out, sharply margined, circular or oval, or more often running together to form vermicular or polycyclical lesions. They usually occupy the scrotum and neighbouring parts of the buttocks and thighs, being confined always to the convex areas and avoiding the sulci and flexures. In some instances the ulcerations have exposed muscle or bone (Fig. 17).

Ætiology and pathology.—The distribution of these eruptions upon the prominent convex surfaces suggests very strongly that they are due to local irritation by wet or soiled napkins, though there is almost certainly another factor which renders them susceptible, namely, some form of gastro-intestinal toxæmia. It is not unusual to find these eruptions associated with Lichen urticatus upon other parts, an eruption which is also probably dependent upon disturbance of the digestive functions. Many of these infants are ill-nourished, but this is not always the case.

FIG. 17.



Diagnosis.—The diagnosis of these eruptions *from those of congenital syphilis* generally demands a good deal of care. The erosive, the post-erosive, and the ulcerating forms are especially liable to be mistaken for syphilis. The features which distinguish them may be summarised as follows:

(1) The "simple erythemas" are limited in a most striking manner to the convex parts of the buttocks, thighs and genitals, and, in extensive cases, of the lower abdomen, calves and heels.

(2) Lesions are absent from the face and from the palms and soles.

(3) They have not the characteristic coppery colour of syphilitic eruptions.

(4) Snuffles, fissures of the lips, hoarse cry, opacity of the skin, all signs of congenital syphilis, are absent.

(5) The course of the eruption is uneven; it does not gradually advance as does a syphilitic eruption, but it varies from day to day, and it disappears on removal of the conditions of local irritation and regulation of the digestive functions, and without mercurial treatment.

II. SEBORRHŒIC DERMATITIS.

There is another distinct and characteristic eruption of the napkin region which is far from uncommon, but which has not been described hitherto as particularly affecting the napkin region of infants in the way that I shall presently detail. This eruption is often mistaken for congenital syphilis.

One may call it "seborrhœic dermatitis" because of the resemblance of its clinical characters to the eruptions which Unna has named "Eczema seborrhœicum." It is to be regarded as distinctly a parasitic (micrococcic) dermatitis, and not as an eczema nor as a seborrhœa. Its characters are as follows: The eruption occupies the whole area covered by the napkin. It forms a uniform bright red sheet, with a granular looking surface from the presence of small moist or greasy yellowish scales. The margins of the area are very sharply defined. In the actual flexures the surface of the eruption may be distinctly moist or oozing. Beyond the margins of the main area there are small pin-head-sized, red, scaly papules, sometimes here and there running together into small patches.

But the eruption is not confined to the napkin region. Other sulci and flexures are involved; as behind the knees, around the umbilicus, in the axillæ, on the neck, behind the ears, and in the labio-nasal sulci. Moreover, the scalp is invariably the seat of a red scaly eruption at the vertex, either as one large patch or in smaller patches (Fig. 18).

Yet another point of importance is that the mother is always the subject of more or less marked "seborrhœa capitis."

The eruption is seen always in well-nourished and apparently otherwise healthy babies with clear complexion. It is very readily removed in a few days or weeks by the application of a mild sulphur ointment, or of some other simple parasiticide such as boracic acid ointment.

Ætiology.—It seems probable that in these cases the baby is infected from the scalp of the mother, and the characters of the eruption and its behaviour under treatment strongly suggest a microbic origin—possibly, as Sabouraud believes for seborrhœa capitis, the *Staphylococcus epidermidis albus.*

Diagnosis.—The features enumerated easily distinguish the eruption *from that of syphilis* and from simple erythema of infants. Some points to be especially noted in differentiating it *from syphilis* are: (1) The healthy clear complexion; (2) the bright red colour and granular surface of the patches; (3) the outlying scale-topped papules (the primary lesions); (4) the particular involvement of the flexures.

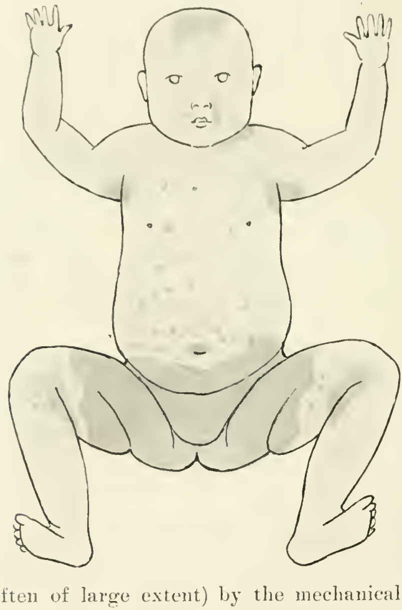
From erythema of infants it is distinguished by its situation in the flexures, the large areas with sharp margin covering flexures and prominences alike, and its occurrence on the scalp, face, axilla, etc.

From impetigo the diagnosis is not always easy, nor, on the other hand, important; but the granular surface, the scaly papules, and the absence of phlyctenules and crusts help to distinguish it.

III. Streptococcic Impetigo about the Napkin Region.

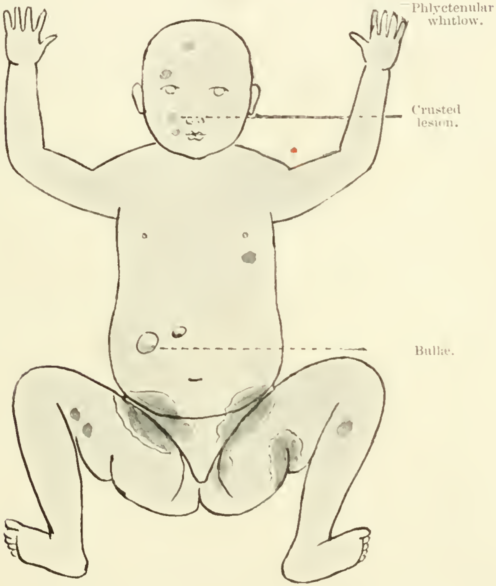
Impetigo contagiosa (phlyctenular or streptococcic impetigo) is prone in young infants to form lesions which retain their phlyctenular character instead of drying up rapidly into the amber crusts so characteristic of the affection in older children. It tends also to attack, not mainly the face, as in older children, but covered parts as well, and especially the napkin-covered areas and the flexures; and in these parts the lesions form excoriated areas (often of large extent) by the mechanical rubbing off of the roof of the phlyctenule. The most severe form of this phlyctenular impetigo is that seen in new-born babies as the result of infection at birth, and known as pemphigus neonatorum. In these cases the eruption is often particularly abundant about the napkin region and flexures, where it may form large raw red areas, but with margins showing the remains of the bullæ.

FIG. 18.



In older infants suffering from impetigo contagiosa there is the same tendency of the eruption to attack the napkin region, sometimes in the form of a large red raw area with phlyctenular margins, in other cases limited to the groins and the mid-thigh sulcus (one form of so-called "intertrigo"). The nature of these excoriated areas is revealed by the phlyctenular margin, and by the presence elsewhere of bullous lesions, and of the more typical amber-crusted lesions of impetigo contagiosa. Often there is a phlyctenular whitlow around a nail on a finger or toe. And other children in the same family may be afflicted with impetigo (Fig. 19).

FIG. 19.



Diagnosis.—(*a*) In new-born babies the simple streptococcic bullous impetigo or pemphigus neonatorum must be distinguished *from the bullous syphilide* which is seen (though rarely) at this period. The impetiginous eruption appears a few days after birth; it may often be traced to infection from the nurse or mother; the child is usually well-nourished, the bullæ are tense. In the syphilitic eruption the palms and soles are especially involved; the bullæ are ill-formed, and often merely raw surfaces covered with brownish shrivelled epidermis, and intermixed with copper-coloured macules. The infant is born with the eruption, and is always much wasted.

(*b*) In infants of a few weeks to a few months old the eruption of impetigo about the buttocks may be *confused with those of syphilis.* The points of distinction are: the absence of coppery tint; the phlyctenular margins; the presence of amber-crusted lesions elsewhere; the involvement of the flexures especially, and particularly the area behind the ears; and the absence of copper-coloured macules on the palms and soles or elsewhere.

Vacciniform eruption about the genitals and thighs in infants.—Here must be mentioned a rare form of eruption, probably of impetiginous (streptococcic) nature, which is seen about the genitals in infants. Primarily the lesions are not very large (pea-sized to finger-nail sized) tense bullæ, few in number, and situated about the genitals and perineum. The bullæ quickly rupture, and excoriated discs or sharply-margined circular ulcers are formed which, unless one is familiar with the eruption, are sure to be mistaken for syphilitic sores. But the children show no other signs of syphilis, the lesions are without infiltration and there are no coppery macules. The result of local treatment proves that they are merely local infections. [For an account of these cases, with a series of excellent drawings, see a paper by Dr. T. Colcott Fox in the 'British Journal of Dermatology,' vol. xix, June, 1907, p. 191.]

IV. THE ERUPTIONS OF CONGENITAL SYPHILIS.

In exceptional cases syphilitic infants are born with a bullous eruption, the so-called "syphilitic pemphigus," and these infants nearly always die. In the large majority of cases, however, the symptoms of syphilis do not appear until the child is from four to eight weeks old. Until that time they may be well nourished and apparently healthy. Then there may appear certain characteristic symptoms: snuffles, hoarse cry, fissured lips and eruptions on the skin. The skin eruptions have certain favourite sites, namely, around the mouth and nose, upon the palms and soles, and particularly about the genitals and buttocks and thighs, *i. e.* in the napkin region.

By far the most common type of skin eruption is that in the form of disc-like patches of coppery red tint. These patches may be erythematous and leaving only a brownish stain after pressure; or there may be some degree of cell-infiltration, so that a maculo-papule results; or the papule may become scaly. Very rarely there are ill-formed vesicles or pustules upon some of the patches.

It is characteristic of these disc-like lesions that they tend to be uniform in size and to attack all parts, flexures and prominences alike. They are generally most abundant over the buttocks, thighs, perinæum, genitals, and lower abdomen, less abundant upon the trunk, and more marked again upon the palms and soles and about the nose and mouth (Fig. 20).

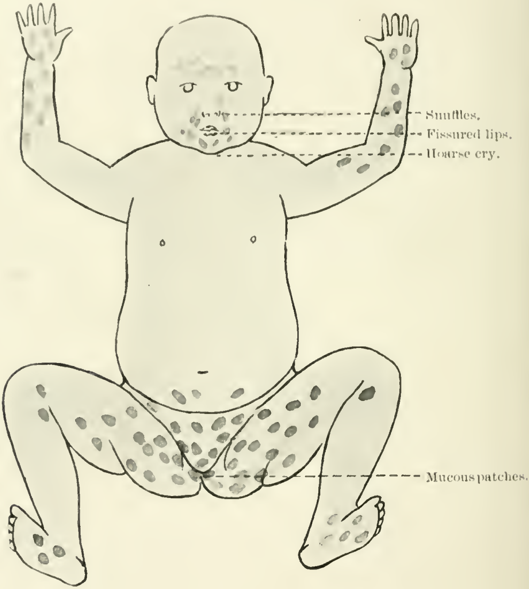
Those lesions which occur about the anus, or in moist flexures, or at the angles of the mouth, often become excoriated and sodden, producing mucous patches or shallow ulcers.

On the thighs and buttocks and around the mouth the disc-like lesions may often run together into large unbroken areas; but always at the margin and on other parts there are to be found isolated macules or papulo-macules.

In my own experience ulcerated lesions upon the skin

—apart from those which may occur in moist areas—are very rare indeed, and most of the ulcerating eruptions in infants which I have seen diagnosed as congenital syphilis have in my opinion been of non-specific nature, either impetiginous or the ulcerative stage of simple erythemas.

FIG. 20.



Diagnosis.—The diagnosis of the eruptions of congenital syphilis from other napkin-area eruptions has already been referred to, and it will be sufficient here to summarise the characters of the specific eruptions:

(1) Appearance of eruption at about the age of four weeks to eight weeks.

(2) Eruption consisting of disc-like coppery-red macules,

maculo-papules, scaly papules, or, rarely, bullous or crusted.

(3) Eruptions situated about the genitals, buttocks, thighs, and often around the mouth, and on the palms and soles, and invading flexures and convex surfaces alike.

(4) Other signs of syphilis: the child begins to lose its plump and healthy appearance, the skin becoming opaque and muddy looking; snuffles, hoarse cry, fissures of the lips, iritis (rarely), enlarged testicle (rarely). The "syphilitic wig" and "old man appearance" are not characteristic signs of syphilis, but may be seen in any condition of malnutrition.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the 13th*, 1907.)

32. "INHERITED SYPHILIS."

By LEONARD GUTHRIE, M.D.

I REGRET that I have no original observations on this subject to bring before the Society. I shall only attempt to discuss whether we are justified in attributing certain forms of nephritis in infants and young children to syphilis exclusively.

The renal conditions which have been regarded as syphilitic are mainly those of an interstitial nephritis with a varying degree of parenchymatous changes.

In recent cases the kidneys may present no morbid appearance to the naked eye, but on microscopical examination the stroma is found to be infiltrated with small cells in scattered areas, together with formation of new connective tissue. The small arteries in the neighbourhood of the glomeruli may be thickened. There may be overnucleation of the glomeruli themselves. Catarrhal changes may be found in the tubules, which may be blocked by hyaline or epithelial *débris*. Small hæmorrhages may be scattered throughout the organs.

In more advanced stages the kidneys have all the appearances of the granular kidneys seen in adult life. They are distorted and shapeless, often of unequal size. The pelvis is dilated, the capsule is adherent, the cortex thin, the glomerular capsules and blood-vessel walls are thickened, the glomeruli themselves atrophied, the tubules obliterated or dilated in cystic form. Signs of recent interstitial or parenchymatous inflammation grafted on to more chronic conditions are frequent.

I know no instances in infants in which gummata have been found in the kidneys associated with the changes mentioned, but if the signs alone of acute and chronic interstitial nephritis in childhood are regarded as syphilitic, it follows that the precisely similar conditions met with in adults must be syphilitic too.

This is a view which I have long been inclined to take, but I must admit that it is non-proven, and lacks sufficient evidence to support it.

I may allude first to cases of nephritis in children undoubtedly syphilitic, several of which have been brought to the notice of this Society already.

Massalongo, of Verona, reported the case of an undoubtedly syphilitic infant which died, aged 8 months, of uræmia. The kidney changes found after death were characteristic of chronic interstitial nephritis in adults.

Coupland and Stroebe have described cases of acute interstitial nephritis in congenital syphilis.

Speiss collected thirty-four cases of congenital syphilis and found that ten had renal disease.

Dr. George Carpenter has related at our meetings the history of two syphilitic and dropsical infants, one aged 5 weeks and the other 5 months. In the one catarrhal changes in the kidneys, with excess of nucleation of the glomeruli and thickening of small blood-vessels were found.

In the younger infant there were no other signs of congenital syphilis, but he regarded the chronic interstitial nephritis present as "doubtless of syphilitic origin."

Dr. Sutherland and Mr. Thomson Walker have also brought before us two cases of syphilitic infants, aged respectively 8 and 12 months. In both marked interstitial changes with catarrhal exudation in the tubules were found, as well as syphilitic endarteritis.

In neither of these cases was dropsy present.

On the other hand, Dr. Ashby has mentioned here an infant which died aged 4 weeks. Anasarca was present from birth, and after death extensive epithelial and fibroid changes, with thickening of small arteries, were noted in the kidneys. There were no signs of congenital syphilis in this case, but Dr. Carpenter thought that the histological appearances supported syphilis.

Emmett Holt collected twenty-three cases of primary nephritis in infants aged from 2½ months to 2 years.

Eleven of these were fatal. Ten post-mortem examinations were held. Five of them showed ordinary parenchymatous nephritis, and five acute interstitial inflammation characterised by small-cell infiltration of the renal stroma and formation of new connective tissue.

Holt does not suggest that any of these patients suffered from congenital syphilis, and it is hardly probable that he would have overlooked evidence of that disease had it been present.

If we turn to children who have died of more chronic forms of interstitial nephritis we must again admit that the evidence of congenital syphilis is far from conclusive.

Dr. James Sawyer, of Birmingham, has carefully analysed twenty-four cases of chronic interstitial nephritis in children and young adults, including seven cases previously recorded by myself.

Two of the number were aged 18 years and the ages of the rest varied from $2\frac{5}{12}$ths to 14 years.

In only one, a girl, aged 18 years, positive evidence of syphilis, in the shape of thirty gummata in the liver and one in the spleen, was found. The syphilis was unlikely to have been acquired as the girl had always been delicate from birth and was stunted and undeveloped at the time of death.

Presumptive evidence of syphilis was afforded in one case by the fact that the father was certainly syphilitic. In another, my own case, the only evidence of syphilis was in the fact that the mother had had six miscarriages, and the patient had the recognised appearance and facies of hereditary syphilis.

In the remainder, syphilis is expressly denied in seven cases and not mentioned in thirteen.

I think we can only fairly conclude that although in a certain proportion of cases of acute and chronic interstitial nephritis in infancy and childhood a syphilitic origin cannot be disputed, that there are yet cases in which similar conditions may proceed from other causes.

I am, of course, excluding cases in which a scarlatinal origin has been found—and also those of calculus.

On the other hand, the well-known difficulty in obtaining a history of syphilis in the parent may account for a number of cases in which the causation of the nephritis remains obscure.

I may mention an experience of my own in point. Some years ago I saw in consultation with Dr. Hugh Armstead an infant aged 6 months. Three months previously it had suffered from iritis of the right eye which was regarded as syphilitic without doubt, although no other signs of syphilis had been present. The iritis cleared up under mercurial treatment, but recently wasting had occurred and incessant vomiting. The urine was scanty and albuminous, containing granular and hyaline and blood casts, and the child died of uræmia a few days later. There was no dropsy at any time. A post-mortem examination was not allowed.

The father positively denied that he had ever suffered from syphilis, and there was nothing pointing to syphilis in the mother or in another healthy child. The mother had had no miscarriages.

At the time I was disposed to doubt the father's word, but possibly I did him an injustice.

But if such cases are not syphilitic, how else can they be explained?

It is possible that some congenital cases may be due to the passage of micro-organisms *via* the placenta into the fœtal circulation.

Cases of congenital jaundice with hepatic cirrhosis which seem analogous to those of interstitial nephritis have been thus explained, and it is certain that these are not necessarily syphilitic.

In other cases it is possible that interstitial nephritis may be secondary to septic pyelitis set up by entry of organisms into the ureters. Such organisms possibly make their way from the bowel to the bladder, thence to the kidneys.

Without doubting that in some instances interstitial nephritis in infancy and childhood is due to congenital syphilis, I think that no case should be regarded as so caused unless other manifestations of hereditary syphilis or a history of them are present.

The only practical suggestion as to treatment which I can offer is, that mercury is a dangerous drug to use in cases of nephritis.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the 13th*, 1907.)

33. "INHERITED SYPHILIS."

BY G. H. TUBBY, M.S.

APART from the "triad" of Hutchinson, namely, auditory, ocular, and dental affections, the bone and joint complications in hereditary syphilis, if not always the most obvious, are certainly the most persistent in their appearance.

The osseous complications are very frequently seen in the cranium, face, and in the extremities. They occasionally occur in the clavicle, ribs, and sternum, but I have not met with the disease affecting the spine.

The cranium.—We are all well acquainted with Parrot's nodes. Extended observation and carefully recorded cases show that the cranial signs may be classified as: (1) Partial or localised, and affecting a segment of the cranium; and (2) general affections of the cranium.

(1) *Partial.*—Partial manifestations are seen on the frontal, the lateral, and posterior parts of the skull, and are frequently associated either with premature closure, or the opposite condition of delayed closure of the fontanelles. The partial manifestations take the form of bosses, which are most marked in the frontal bones. According to Edouard Fournier hereditary syphilis may manifest itself, so far as the frontal bones are concerned, in three ways. The whole of the frontal bone may be unduly convex and prominent, giving rise to what is termed in France the *front Olympien,* or as it is sometimes called in this country the belly-like frontal bone. The more common condition of affection of frontal bones is an exaggeration of the normal eminences; and a rare form is the keel-shaped frontal bone, in which, in the position of the interfrontal suture, a vertical ridge of bone 1–2 cm. in breadth is developed, while the lateral aspects of the bones are flattened.

The local lateral manifestations of the disease are seen in the familiar enlargements of the parietal eminences, which may exist alone, or in conjunction with enlargement of the frontal eminences. In the latter form case we see the characteristic natiform or "hot-cross bun" cranium.

The rarest of all forms of partial manifestation of hereditary syphilis of the cranium is the occipital enlargement. This takes the form of an excessive overgrowth of the occipital protuberance.

As to the histology of these hypertrophies, space does not permit me to refer to it.

(2) *General.*—This may take the form either of increase or decrease of the normal size of the skull-cap. The globular cranium associated with hydrocephalus is a type of the increased form, and is often difficult to diagnose from rickets with hydrocephalus. In hereditary specific disease it is found that most of the children affected die young, and do not survive until the usual time of the advent of rachitis. Characteristic examples of general decrease in the size of the cranium are seen in some microcephalic children. And lastly, Fournier has noticed the constant association of cranial asymmetry with inherited disease.

Of the bones of the face, the nose shows the most characteristic changes. In occasional instances the root of the nose is broadened extremely, and gives to the patient a leonine aspect. In other cases the course of the disease is destructive, and the bones of the nose become affected secondarily after the onset of chronic coryza, followed by purulent discharge, epistaxis, and ozæna. The type of nose seen varies. In some cases the depression is more marked at the bridge, while in other cases destruction has taken place at the extremities of the nasal bones, and there has also been some loss of the cartilage. Hence the nose is tip-tilted, and there is a deep furrow extending across it at the junction of the cartilage and bone.

With regard to the bones, even beyond the age of puberty, patients suffering from hereditary syphilis suffer from chronic pains, which are frequently ascribed either to growing pains or to rheumatism. The pains are felt more often in the legs than in the arms, and particularly about the crest of the tibia. They are exaggerated at night, and are sufficiently severe to keep the patient awake. They often come on in paroxysms, in which a distinct crisis can be recognised. In many instances they are not associated with any external signs, but in other cases distinct evidences of chronic osteitis, hypertrophy of large bones and gummata are found. The pains often last months or years, and not infrequently their nature escapes diagnosis.

The most striking of all the lesions of hereditary syphilis is what is known in France as the *Maladie de Parrot*, or as we term it here "syphilitic pseudo-paralysis." It occurs in young infants, and before the fourth month, and is most common during the second month of life. It comes on abruptly without any apparent cause. There is no history of injury, but the limb becomes useless and limp; hence the title "pseudo-paralysis." Then a swelling develops in the juxta-epiphysial region, which is tender on pressure, and associated with it there is an effusion into the neighbouring joint. If the disease be left untreated, separation of the epiphysis occurs and spontaneous fracture follows. In some instances pus forms, and the entire epiphysis is necrosed, and may require removal. But it is striking to note the effect of mercury in these cases, even when the disease has progressed beyond the serous stage of inflammation. If the pus be let out early and mercury vigorously administered, it often happens that the epiphysis is saved and may re-unite.

With reference to the other bone lesions, they are classified as—(1) localised thickenings, taking the form of hyperostoses or exostoses; (2) cicatrices adherent to bone; and (3) local deformities.

The exostoses are found either in the epiphyses or the diaphyses. The epiphysial variety most frequently affects the upper end of the tibia, but it is also seen in the extremities of the radius, the ulna, and at the malleoli. The diaphysial type of bone lesion is best marked in the tibia and the phalanges. In the tibia the exostosis or hyperostosis may be localised, and break down in the form of gummata. Or the whole shaft of the bone may be affected, giving rise to a perfectly characteristic deformity, which the French call *tibia en lame de sabre.* It is often mistaken for rickets, but the following points of distinction should be noticed: The curvature in specific disease is, as a rule, directly anterior, whereas in rickets it is mostly antero-lateral. In specific disease the crest of the tibia is rounded; in rickets it is sharpened. In specific disease the external and internal surfaces of the bone are convex; in rickets they are flattened and concave. As to the age when the deformity appears, in syphilis it may come on at any time up to the fifteenth year, in rickets rarely beyond the third year. In place of the coarse swellings and hypertrophies of the bones, their surfaces may be affected by numberless little irregularities. Many of these bone affections are associated with suppuration and give rise to cicatrices.

Joints.—Affections of these structures are usually secondary to those of the bones in the neighbourhood. In some cases of syphilitic epiphysitis the inflammation extends to the joint, and results in simple serous effusion or hydrarthrosis. In other cases the disease may be so extensive as to seriously involve the joint, either by direct continuity or by perforation and irruption of pus into the joint cavity. In older children, particularly about the ages of twelve to fourteen, a rarefying osteitis, associated with a deposit of caseous material in the cancellous tissue, affects many bones. The process extends to the articular cartilages, and results in grooving and pitting, the condition being known as chondro-arthritis. The signs of the affection are recurring attacks of synovitis, with chronic inflammation of the neighbouring bone. This form of disease is said not to respond to the usual remedies, and is practically incurable, in fact it may be described as a para-syphilitic manifestation.

Gummatous thickening of the synovial and sub-synovial tissues is not uncommon, and is usually associated with osteo-periostitis with the formation of osteophytes. Partial loss of movement and even complete ankylosis results, with muscular and ligamentous contractions.

It is noteworthy that the manifestations above referred to are usually seen at the larger joints, particularly the shoulder, elbow and knees. It is a question whether some forms of chronic deforming joint disease, having a very close resemblance to chronic deforming rheumatism and occurring in young people, are not manifestations of hereditary disease. It is certain that if careful inquiry be made, some of these children show unequivocal signs of syphilis.

But while the general surgeon, on the one hand, is apt to overlook the more remote sequelæ of the affection, and often fails to trace conditions to their origin; on the other hand, syphilographers are prone to assume that any disease occurring in a patient congenitally afflicted must necessarily be specific in origin, and some of the inferences and statements of these latter observers must be received with considerable caution and even scepticism.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the* 13*th*, 1907.)

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34. "INHERITED SYPHILIS."

By GEORGE CARPENTER, M.D.

The subject which has been selected for discussion to-night presents diagnostic problems of interest to the clinician, for although in many instances the nature of the complaint we are called upon to treat is only too obvious, there are other cases where the diagnosis is neither easy nor certain.

It is with these latter examples occurring in infants that I propose to deal along with other matters in regard to congenital lues which have attracted my attention from time to time.

Chronic "Snuffles."

The clinical significance of chronic "snuffles" in certain of its phases is not devoid of interest. The infant suffering from chronic "snuffles" with a muco-purulent discharge bubbling from its anterior nares is recognised by all, but I don't think it is sufficiently understood that there may also be chronic "snuffles" localised to the posterior parts of the nasal cavities and to the naso-pharynx, attended by a considerable muco-purulent discharge which finds its way into the infant's stomach by the posterior nares, and which, by reason of its situation, is likely to escape notice. I have elsewhere remarked on this, and Paul Gaston * has confirmed these observations, and he has also called attention to the dangers of streptococcal infection from that source. He attributes the common causes of death, viz. broncho-pneumonia, diarrhœa and vomiting, and marasmus, to the direct transference of streptococci and to the absorption of their toxines, and also, in some instances, to the invasion of the blood-stream by these and other germs.

These and others of the complications that Gaston assigns to streptococci can, in the light of modern discovery, be as well, if not better, explained by the action of the specific germs. But as a contributory cause of death streptococcal infection cannot be overlooked.

The recognition of the concealed variety of "snuffles" is not only important for diagnostic purposes, but its presence must not be overlooked for therapeutic reasons. The local treatment of chronic "snuffles" by nasal applications is most necessary. Infants with blocked nostrils cannot take sufficient nourishment or properly inflate their lungs, and the dangers of sepsis from the nose and naso-pharynx are by no means to be ignored. But I must confess that some cases of chronic "snuffles" with copious muco-purulent discharge are difficult to place. They are apparently not syphilitic, and they do not yield to anti-syphilitic medication. They are not due to post-nasal growths (adenoids). But what they are due to is not clear. An example of this condition is a case I showed to the Society this session, No. 67, page 292.

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* 'Revue d'Hygiène et de Médecine Infantiles,' tome 3, No. 3, 1904.

CRANIO-TABES.

I propose next to offer a few observations on cranio-tabes and Parrot's nodes. There are certain inaccurate statements in regard to one of these conditions which still appear in the text-books, although it is many years now since I first drew attention to the inaccuracy of that doctrine.* I refer to the situation of the lesions of the former and to the time of their occurrence. On these

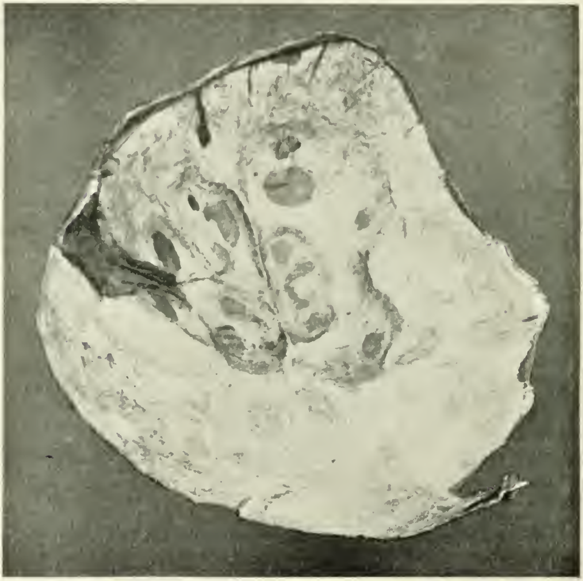

FIG. 21.—Cranio-tabes of left parietal bone. In many places the bone has disappeared, leaving a membranous covering only. Note the atrophied areas on the inner table showing as pits and depressions in the bone. (Photo. by Dr. A. W. Harvey.)

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* "Cranio-tabes in Young Children: A Clinical Inquiry into its Origin, Illustrated by 100 Cases," 'St. Thomas's Hospital Reports,' 1889.

points—situation and time—there should be no disagreements, whatever views may be held in regard to causation.

The common situation for cranio-tabes is behind the parietal eminences, and in 95 per cent. of the cranio-tabic cases the parietal bones are attacked. In 60 per cent. the parietal bones are solely involved, and in less than 3 per cent. the occipital bones. Other cranial bones are occasionally involved, but I have never seen the frontal bone alone affected.

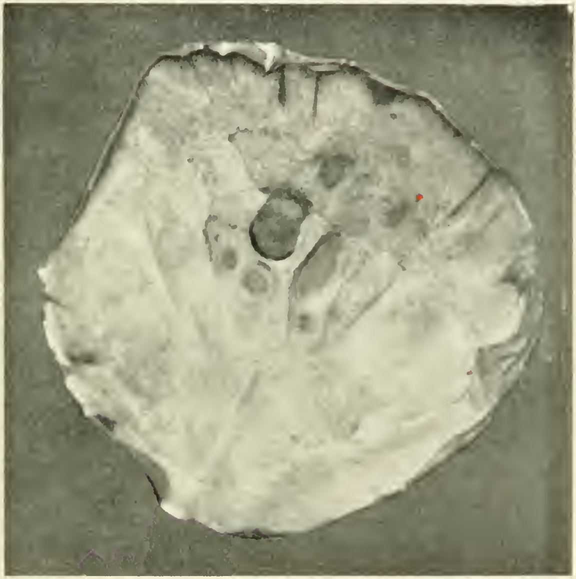

Fig. 22.—Cranio-tabes of right parietal bone. In several places the bone has disappeared, leaving a membranous covering only. Note the atrophied areas on the inner table showing as pits and depressions in the bone. (Photo. by Dr. A. W. Harvey.)

Another point of interest is that cranio-tabes is most often found in the second and third months of life, and that after six months of age the decrease in the number of the cases is most striking. In other words, in what may be termed the *syphilitic age* cranio-tabes is quite a common occurrence, while in the *rickety age*, that is, from the ninth month of life onwards, the reverse is the case.

There are certain observations the truth of which cannot be denied, viz. that cranio-tabes is found in syphilitic fœtuses, and that it occurs in syphilitic infants.

I have found the condition as an isolated lesion in cases where I have known one or other parent to be syphilitic; as an isolated lesion where I have attended a brother or a sister immediately preceding the patient for syphilis; also in association with marked anæmia which rapidly responded to mercurial treatment; and also associated with chronic "snuffles"—quite a common combination.

There are other points about cranio-tabes in relation to this question of syphilis that I think ought to be emphasised. Some cases recover quickly under mercurial administration, but others less readily, and in others this treatment is of no value.

I have no doubt that cranio-tabes is produced by syphilis during the *syphilitic age*, and that it is just as much a symptom of the complaint as an enlarged liver and a swollen spleen, and that, like these manifestations, it is not pathognomonic. In quite a number of cases examined by the usual clinical methods there is not a suspicion of syphilis, and it remains to be proved whether any or all of these cases will be found to be of a syphilitic nature by future bacteriological and cytological investigations. We have been brought to a standstill at this point, and we need the additional information which such examinations would afford to settle the question.

The next point for consideration is that the more rickety the infant the less likely is it to be cranio-tabic. Cranio-tabes does occur in the *rickety age*, there is no doubt about that also, but it happens infrequently. Rickets, on the other hand, is a very common malady indeed.

But although syphilis is a disorder which is at its height during the first six months of life, it is by no means wiped out in the succeeding six months and obvious sporadic cases of congenital syphilis are met with in the *rickety age*.

There is sufficient objective evidence, therefore, to satisfy the critic that syphilis is still in operation although its recognised manifestations are apt to be infrequently seen.

In some infants cranio-tabes disappears and rickets persists or even increases. Further, I have seen cranio-tabes recover and give place to Parrot's nodes.*

Since Elsässer first described the condition in 1843 and attributed it to rickets and since Parrot challenged the rachitic explanation there has been a see-saw of opinions, some believing in its rickety origin, others in its syphilitic, while others, again, pinned their faith on the combined explanation.

But I have no confidence in its rachitic origin for the reasons I have just advanced.

Whenever I find cranio-tabes in an infant I *suspect* syphilis, but I must admit that my suspicions are not always verified by the test of mercurial treatment. Indeed, some of the infants recover without mercurial treatment. But the above suspicion is a good working hypothesis.

PARROT'S NODES.

Unlike cranio-tabes Parrot's nodes is met with in the *rickety age*, not the syphilitic.

It occurs when the evidence that can be obtained of syphilis is of the weakest possible kind.

Another point about it is that when it does happen it usually arises in rickety children.

The associated rickets may be of all grades, from a condition of body which cannot be claimed to be free from rickets to one where that disorder is very pronounced. But I have detected it in non-rickety subjects.

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* "A Case of Scurvy in a Cranio-tabic Infant, with Observations on Cranio-tabes,' 'Reports of The Society for the Study of Disease in Children,' vol. iii, pp. 214–222.

Cranio-tabes is not often found with Parrot's nodes, but I have met with this association three times in forty cases.

What I consider to be a striking fact in regard to Parrot's nodes is that although, as I have already pointed out, syphilitic evidence is usually wanting by reason of its time incidence, nevertheless no less than 57·5 per cent. of my cases were undoubtedly syphilitic.*

There is also another fact which I believe I was the first to note, and to which I should like once more to call attention, and that is that splenomegaly is a frequent accompaniment. Half of my cases of Parrot's nodes were associated with enlargement of the spleen.

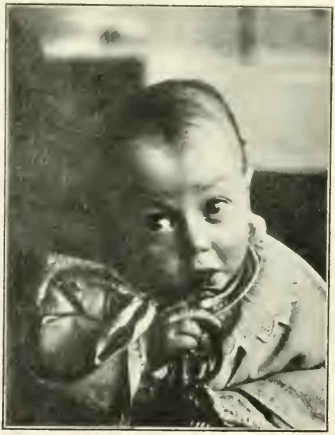

FIG. 23.—The "hot cross bun" variety of Parrot's nodes.

There is one other observation already published by me elsewhere, and that is that one of these patients was the child of a woman suffering from congenital syphilis. It is the only example I have met with of possible transmission to the third generation.

In my opinion Parrot's nodes is a syphilitic manifestation and not a rickety disorder. It is allied to the periostitis which occurs in the long bones of infantile syphilitics, and which may either be found in association with epiphysitis (osteo-chondritis) or without that condition. I have met with typical Parrot's nodes in children suffering from periostitis of the long bones.*

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* 'Syphilis of Children,' p. 73.

The usual situation for cranial osteophytes is in the neighbourhood of the anterior fontanelle. When in front of it the characteristic buffer-like bossing of the frontal bones is produced (Fig. 23). If combined with bossing of the parietal bones behind the anterior fontanelle there results the hot-cross-bun skull with its cross over the suture lines (Fig. 23). Intensification of the natural anatomical protuberances is the rule, but no part of the skull is exempt. In its earliest and least characteristic stages sharply circumscribed osteophytes, ranging from a pin's head to a half-crown or more in circumference, may be scattered over the vertex. The characteristic lesions and the small disseminated nodes may occur together. In its later manifestations characteristic formations are apt to flatten down, leaving the cranial vault thickened, an object of suspicion perhaps, nothing more.

Syphilis as a Rickets Producer.

Before leaving this question of syphilis and rickets I should like to say that I have no examples in my case-books of cases of pronounced rickets arising in syphilitic children in the *first six months of life*, nor can I remember to have seen such.

I have seen rickets develop in syphilitic children that have been fed on the *breast alone*, and, therefore, occurring in infants in whom there was *primâ facie* evidence that rickets had been produced by the syphilitic virus only.

But against this assumption is the undoubted fact that rickets does occur in non-syphilitic breast-fed children where lactation has been unduly prolonged.

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* *Loc. cit.*, p. 74; 'Reports of The Society for the Study of Disease in Children,' vol. iv, 1904, p. 159.

Cases of beaded ribs I have seen in non-syphilitic infants, but beaded ribs are not special to the syphilitic. Besides, beaded ribs occur in infants at birth, and the older I become the less impressed am I by slight enlargements of the costo-chondral junctions. If every infant who displays a ridge at the costo-chondral junctions is to be considered to be possessed of an incipient rickety rosary, and is to be viewed as an early example of rickets, then a non-rickety infant is a great rarity.

I have hitherto looked upon syphilis as a producer of rickets, but I cannot persuade myself that I have had any sound clinical justification for doing so. Syphilitic infants, like other children, are apt to become rickety if improperly fed and environed, but it does not strike me that they are more liable to rickets than the non-syphilitic or sick children in general.

But it is always well to hear the other side, and the very latest pronouncement on these debatable questions is by Marfan.* He thinks it would be a mistake to deny syphilis any share in the ætiology of rickets. He holds that syphilis is by itself sufficient to produce rickets, and is not merely a predisposing cause as Fournier thinks. The peculiar characters of syphilitic rickets are—(1) its early appearance; it is either congenital or starts in the first three or four months of life; (2) it is remarkable for its cranial lesions, especially cranio-tabes and the natiform skull, which are both much commoner in syphilitic rickets than in rickets due to other causes; (3) marked anæmia; (4) chronic hypertrophy of the spleen, which, in two thirds of the cases, is due to hereditary syphilis. Rickets due to alimentary disturbance hardly ever shows itself before the sixth month, is not well marked before the second year, affects the limbs more than the skull, and is often associated with digestive troubles and a big belly. Anæmia is less marked, and hypertrophy of the spleen is infinitely less frequent than in syphilitic rickets.

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* "Rickets and Syphilis," 'Semaine Med.,' October the 2nd, 1907, p. 469.

Syphilitic Epiphysitis.

In only one case, and that in a syphilitic infant, aged 2 or 3 months, have I seen a pronounced enlargement of the costo-chondral junctions, and it is more than likely that that was osteo-chondritis, and not rickets.

If there be no periosteal extension up the shafts of the bones syphilitic epiphysitis is much like a rickety enlargement.

Sometimes the collar of enlargement between the epiphysis and the diaphysis does not extend all round the bone end. Then there is no doubt about the diagnosis, and there is no doubt if there be a condition of pseudo-paralysis; and there is also no doubt about the diagnosis when the epiphysis and the diaphysis part company, as sometimes happens.

The bulk of the cases of epiphysitis that I have met with have occurred during the first three months of life, but I have seen examples in the *rickety age.*

Thus in one child, aged 17 months—the oldest I have seen—both elbows were involved.

In a child of seven months both knees and elbows were attacked, and there were tibial nodes also, and in another of the same age both knees were affected.

It has not been unusual* in my experience to find multiple epiphysitis as the sole manifestation of hereditary syphilis. Of 41 epiphyses involved there were 15 elbows, 11 wrists, 10 knees, 3 shoulders, and 2 ankles. In one case there was myositis, the neighbouring affected muscles being hard and painful to touch. Chapin and Henoch also have recorded similar experiences. But any lesions of the muscles have been in my practice very uncommon, and I can only call to mind once having seen gummata which were in the amputated thigh muscles of a syphilitic child.

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* "A Case of Syphilitic Epiphysitis," 'Reports of The Society for the Study of Disease in Children,' vol. i, p. 16; 'Syphilis of Children,' p. 66.

SYPHILITIC SPLENOMEGALY.

The spleen is commonly enlarged in syphilitic fœtuses, and of all organs next to the liver it is the most prone to suffer in intra-uterine life. Indeed, according to many writers it is the organ most usually involved in extra- as well as intra-uterine life.

Referring for the moment to splenic enlargements in general in infancy and early childhood, there are two periods of that life when it is most usual to find splenic enlargements, viz. during the first six months and from the first to the third year, or a little longer. Between the sixth and the twelfth month there is a lull in splenic excitability. It is during the first three years of childhood that the finding of an enlargement of the spleen may be looked upon as nothing out of the way, but after that age splenomegaly is uncommon, and becomes increasingly so as the child gains in years.

This age comprises two phases of child-life, viz. what I have termed the *syphilitic age* during the first six months, and the *rickety age* from, say, the ninth month to the third year.* Many cases occurring during the first six months of life are unquestionably syphilitic. Various estimates have been made of the frequency of enlargements of the spleen in this disorder. Thus splenomegaly occurred in 40 per cent. of my cases, but other observers give far higher† figures, viz. 62 per cent., with nineteen probabilities in addition according to one of them. These discrepancies are doubtless due to the conceptions formed by individual observers as to what may be regarded as evidence of syphilis.

During the *syphilitic age* rickets is practically non-existent, and therefore any influence it may be supposed to exert can be neglected. But although there is no rickets to confuse the issue, there are doubtless poisons, other than the syphilitic, to account for splenic enlargements.

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* "On Splenomegaly in Infants and Young Children," 'Reports of The Society for the Study of Disease in Children,' vol. iii, pp. 296–320.

† 'Syphilis of Children,' p. 50.

During the *rickety age* splenomegaly is met with once again in increasing frequency. It is during this period of life that obvious syphilitic disorders are much less in evidence, and that splenic enlargements can then with less show of reason be attributed to syphilis. But some of these cases are without question syphilitic.

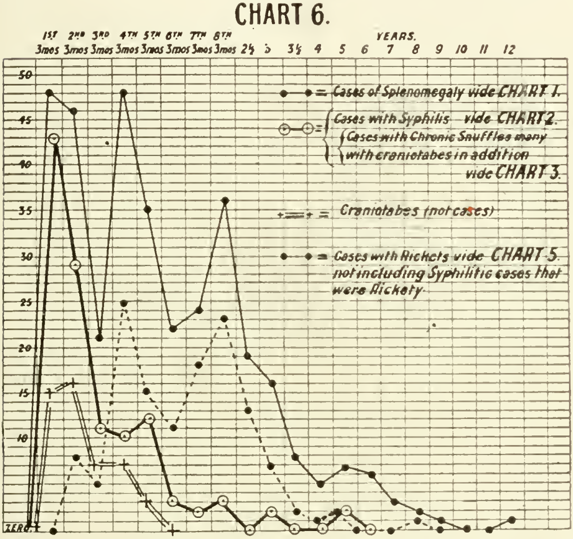

FIG. 24.—Illustrating Dr. George Carpenter's paper on "Splenomegaly," 'Reports of The Society for the Study of Disease in Children,' vol. iii, 1903, pp. 296–317.

In regard to rickets, it is questionable in my mind whether splenic enlargements arising at this age in rickety children are due to rickets. My own conviction is that splenomegaly in this complaint is an epiphenomenon.*

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* *Loc. cit.*, p. 306.

I also hold that in the rickety age also there are other agencies besides syphilis and rickets to account for splenic enlargements at this period of life of which we at present have no knowledge. It is daily brought prominently to our notice that the spleen is very sensitive and readily responds to a variety of *known* infections; thus tubercle, enteric, pneumonia, whooping-cough, and so on will all bring about swelling of that organ, and there must be other *unknown* infections with like spleen-swelling propensities, and this must be the case if only for the reason that the *known* infections are not sufficient to account for all the cases of splenomegaly that arise in infancy and early childhood.

But what in this matter is of real interest to the clinical physician is, what interpretation is to be placed on the doubtful case? What is then the significance of splenomegaly? For instance, the common association of splenomegaly with Parrot's nodes. I certainly view the enlarged spleen in these cases as syphilitic. But there are other associations with splenomegaly, thus, cranio-tabes and chronic "snuffles." Sometimes it is with one symptom, sometimes with the other, sometimes with both of them. If these symptoms occur with anæmia, as sometimes does happen, and if the anæmia and the cranio-tabes respond to the mercurial test, then there can be no question as to the significance of the enlarged spleen. But the, to us, unexpected perhaps happens, the treatment is disappointing, and we are left wondering whether the test of trial by mercury is, after all, competent to decide the question.

How are we in the present state of knowledge to interpret the following clinical riddles, and what bearing, if any, have they on the disease in question? The onset of splenomegaly in already rickety and anæmic children; the onset of splenomegaly in children who have been rickety but who have recovered; diminution in the size of the spleen while the children become more rickety. What is the explanation of such phenomena as the following? Twin sisters with splenomegaly. One, aged 14 months, died of pronounced anæmia (hæmoglobin 18 per cent., colour index ·33, red corpuscles 55 per cent., marked alterations in them, white ratio 1 to 172). The other developed Parrot's nodes, and became decidedly rickety, but was then not anæmic though she had been (hæmoglobin 42 per cent., colour index ·6, red corpuscles 68 per cent.). In another set of twin infant sisters in whom there was evidence neither of rickets nor of syphilis, in one the spleen was enormously enlarged, while in the other it was quite natural.

Soon we may be in a position to solve some of these clinical problems, and perhaps in the doing may expand as well as recast our present ideas of syphilitic disorders. The *Treponema pallidum* * has now been demonstrated in the blood and in all the organs and tissues of the body. Observations are being made on the cerebro-spinal fluid of congenital syphilitics. Jobler † has found characteristic changes there, and we are in hopes of fresh and important developments along these lines of research. But until we are placed in possession of some such aids to diagnosis, the syphilitic origin of some splenic enlargements, as also other doubtful symptoms, must be mainly conjectural. In the meantime no harm will be done when an enlarged spleen is discovered by being suspicious of syphilis, and that, I think, should be the attitude of mind in regard to these and other doubtful cases. A suspicious attitude of mind should make the clinical examination of the child more thorough, not less searching.

Syphilitic Enlargement of the Liver.

Not only are splenic enlargements common during the syphilitic age, but enlargements of the liver follow them fairly closely, if anything being a trifle in excess in my experience.

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* 'Atlas der Ätiologischen und Experimentellen Syphilisfossihung.' Von Erich Hoffmann, Berlin, 1908, 34 plates (30 coloured and 4 photomicrographs).

† "Lymphocytes of the Cerebro-spinal Fluid in Congenital Syphilis and its Diagnostic Significance," 'Jahrbuch f. Kinderheilkunde,' 64, i, 1.

And what has been said about the spleen in regard to congenital syphilis applies equally forcibly to the liver.

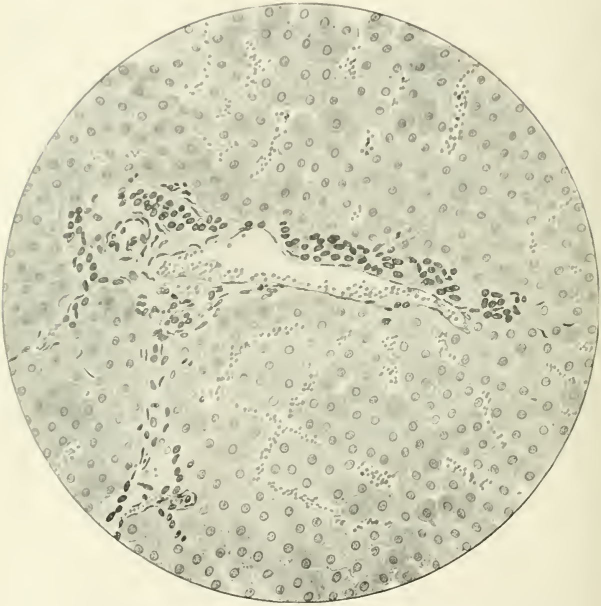

FIG. 25.—Section of liver showing slight leucocytal infiltration of the portal canals. The capillaries contain a quantity of red blood-corpuscles. Leitz oc. 1; obj. 7.

Although it is a common enough occurrence to find enlargement of the liver in syphilitic infants clinically, yet the detection of any pronounced alterations therein by palpation during life or the finding of naked-eye pathological changes after death is not usual.

Sometimes, clinically, the liver feels hard, but ascites or jaundice * are most uncommon.

Diffuse cellular infiltration (interstitial hepatitis), similar to the infiltrations occurring in other organs, and first described by Gubler, is the common mode of attack.

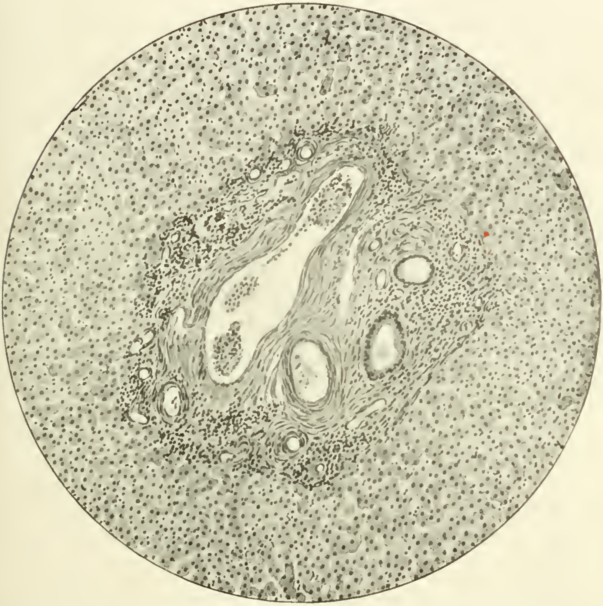

Fig. 26.—Section of liver showing infiltration of a portal canal with leucocytes and invasion of the peripheral liver-cells by leucocytal infiltration. Note that the capillaries of the liver contain large numbers of leucocytes. *Vide* "Syphilitic Nephritis in Infants" (continuation of present article). Specimen taken from same case as Fig. 29. Leitz oc. 5; obj. 4.

In its less marked forms it appears as a slight leucocytal infiltration of the portal canals (*vide* Fig. 25), insinuating itself between the peripheral cells of the neighbouring hepatic lobules up to a very pronounced fibro-cellular invasion of these parts associated with dispersal and destruction of the liver-cells. The vascular walls are infiltrated (Fig. 26). Accompanying this condition there may be naked-eye gummata, even as small as a millet seed (miliary syphilomata of Wagner), or microscopical gummata, and these latter may alone be disseminated throughout the gland. Gummata visible to the naked eye are, however, quite rare in infants. I have only seen the condition once, viz. in an infant, aged 8 months,* in the shape of two small nodules. Coupland,† however, in a child, aged 3 months, found one large orange-yellow gumma, measuring 4 by 2½ in., along with three others the size of beans.

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* 'Syphilis of Children,' p. 54.

Gummata, microscopically, consist of small round cells seated in a fine network of fibrils together with transitional nuclei intermediate between round and spindle shapes.‡ Isolated small gummata cannot, of course, be discovered clinically, but where there is decided portal infiltration the liver shows unmistakable naked-eye alterations. It is large and hard or elastic and india-rubber like. Its edge is rounded. On section it cuts with a creaking noise and sensation, and in colour it is either yellow, or flint-like, or pale.

But indurated livers are rarely encountered clinically; on the other hand, it is reasonable to assume that the enlargements which are so common in this disorder are, in some cases at least, owing to less pronounced changes of the same character.

SYPHILITIC ADENITIS.

I have but few observations to make on syphilitic glandular enlargements. There may be found enlargement of some or all of the accessible lymphatic glands in cases affected with chronic "snuffles" or in those suffering from cranio-tabes in addition.

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* *Loc. cit.*, p. 52.

† 'Trans. Path. Soc.,' vol. xxvii, 1896.

‡ For a coloured plate of this condition see "Étude de 95 Cas de Syphilis Infantile," plate viii, A. Fruhinsholz, 'Revue d'Hygiène et de Médicine Infantiles,' tome ii, No. 1, 1903.

I have not infrequently detected slight enlargements of the epitrochlear glands in syphilitic infants and in those who have excited strong suspicions of that malady. Lancereaux [*] states that the lymph glands of the abdomen are often found enlarged in those infants with splenomegaly.

SYPHILITIC NEPHRITIS IN INFANTS.

Syphilitic nephritis in infants is, I think, of more than passing interest to the physician, not only on account of its immediate danger to life, but owing to its more remote possibilities.

The kidneys, like other organs, have been found diseased in syphilitic fœtuses, and their retarded development has been described by various writers, viz. Stroebe,[†] Stoerk,[‡] and Cassell.[§] Speiss [‖] found post-mortem evidence of renal disease in ten out of thirty-four cases of congenital syphilis, and Lancereaux [¶] stated that he found in a nursling of Tarnier's connective-tissue proliferation with fatty degeneration of the epithelium lining the tubuli uriniferi, the organs being of a firm and yellow colour. Coupland [**] found in a moribund plump female child, aged 3 months, the kidney cortex the seat of an interstitial infiltration of small round cells, most abundant around the arteries and especially around the Malpighian bodies. The renal epithelium was unaltered. The kidneys "to the naked eye presented a normal appearance." Cassel* found definite interstitial changes in the kidneys of five infants who survived for some time after birth, in the form of various sized cellular groups of infiltration in the cortex and cystic degeneration of the glomeruli. In all there was proliferation of the adventitia of the interlobular arteries consisting of polynuclear, mononuclear, eosinophile, and plasma-cells. The process had in no case proceeded to the formation of connective tissue. Of seven specimens taken from syphilitic fœtuses six showed inflammatory interstitial cellular proliferation along with peri-adventitial infiltration. The interstitial changes displayed no features by which they could be distinguished from any other inflammatory process, but the peri-adventitial changes he regarded as of strong diagnostic import.

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[*] 'Annales de Dermatologie et de Syphiligraphie,' No. 4, 1872–3, "Des affections viscerales de la Syphilis Héreditaire," p. 328.

[†] 'Centralbl. f. Allg. Path.,' vol. ii, 1891.

[‡] 'Wien. Klin. Woch.,' 1901, No. 41.

[§] "Nephritis in Hereditary Syphilis," 'Berl. Klin. Woch.,' 1904, No. 2.

[‖] "W. Die Verschiedenen Mereis Affeckionem bei Syphilis Constitutionales," 'Inaug. Diss. Berl.,' 1877; quoted by Stroebe.

[¶] *Loc. cit.*, p. 322.

[**] 'Trans. Path. Soc.,' vol. xxvii, p. 303, 1876.

Massolongo, of Verona,† reported a case of syphilitic nephritis in a child, aged 8 months, who died with uræmic symptoms. The renal arteries presented marked signs of endo- and peri-arteritis, and the same lesions were found in lesser degree in the liver and spleen. There was connective-tissue sclerosis of the kidneys. The histological changes found were similar to those seen in late renal syphilis (chronic interstitial nephritis), in distinction to those of precocious renal syphilis (parenchymatous nephritis, glomerular nephritis). Although in this case the lesions were of intra-uterine development, the anatomical changes were those of the late adult form of renal syphilis.

In a wasted syphilitic infant, aged 8 months, I found a small gumma in the left kidney and two small gummata in the liver.‡

Fruhinsholz,§ in an important study of ninety-five cases of infantile syphilis, remarks that the kidneys of many syphilitic infants were pale and small, but that they did not usually show well-marked naked-eye changes. He gives several illustrations. In a syphilitic infant of a few weeks old there was thrombosis of the renal veins accompanied by hæmorrhage into the tubes and between them. There was complete apoplexy of one kidney and partial apoplexy of the other. In another syphilitic infant a few days old he found thickening of the intima of certain vessels up to complete obliteration, thickening of the capillary walls with amorphous (amyloid ?) substance, and in one microscopical section a large artery was seen with an amorphous outer coat. He also described interstitial fibrosis, fibrous thickening of Bowman's capsules, enormously thickened tubule walls which appeared to have undergone amyloid degeneration, and small and amorphous Malpighian tufts. Within the tubules were desquamated epithelial cells, becoming granular or changed into an amorphous detritus. In another syphilitic infant, aged 5 months, there were diffuse sclerosis, glomerulitis, and fibrous endarteritis. In a wasted infant, aged 9 months, unsuspected of syphilis, he found in one kidney three little round gelatinous gummata tinged with violet and striated with white, varying in size from a hemp-seed to a small pea. In the other kidney was a similar gumma the size of a pea. Microscopically they consisted of round mononucleated, sometimes polynucleated cells, and here and there some fusiform elements. The tubules were thrust aside by the new growth and filled with round and fusiform cells, and atrophied glomeruli were invaded by round cells. Little lakes of extravasated red blood-corpuscles were also seen. Outside the gummata the kidneys were barely altered.

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* *Loc. cit.*

† International Medical Congress, Rome, March 29th—April 5th, 1894; 'New York Medical Record,' April, 1894, p. 534.

‡ 'Syphilis of Children,' p. 52.

§ 'Revue d'Hygiène et de Médicine Infantile,' No. 1, tome ii, pp. 41–47, 1903.

Pathological examples of the disease in infants have also been exhibited at our Society by Sutherland and Thomson Walker.* In a girl, aged 16 months, the subject of syphilitic arteritis of several main cerebral arteries, they found the interstitial tissue of the kidney cortex

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* 'Reports of The Society for the Study of Disease in Children,' vol. iii, 1903, pp. 134–146.

(chiefly) densely infiltrated with round cells and the tubules displayed catarrhal changes.

They subsequently examined, post-mortem, the kidneys of a syphilitic girl, aged 8 months, and found similar microscopical changes to those described in their first case.

But all these cases, valuable as they are as pathological records of infantile syphilis of the kidneys, are wanting in that which is of most interest to us at the present moment, viz. a clinical side.

The distinction, however, of being the first to recognise and successfully treat a case of syphilitic nephritis in an infant belongs to S. Messenger Bradley, of Manchester.* The infant was a girl, aged 4 months, "covered with a syphilitic psoriasis of one week's duration." The face, arms, and legs were œdematous and readily pitted on pressure. The urine contained four fifths albumin and numerous epithelial and granular casts. He ordered hydrargyrum c̄ creta gr. ii *bis die*, and a "little" unguentum hydrargyri to be rubbed into the abdomen each night. In the course of three or four days under the action of the drug "the syphilis began to pale and fade away." "After three weeks' treatment the albumin in the urine and the eruption on the skin had entirely disappeared, the cellular tissue was free from dropsy and the microscope failed to reveal the presence of any abnormal element in the urine."

This instructive clinical observation, however, bore no clinical fruit, for during a period of thirty years no cases were recorded by those specially working in children's diseases, or by others, of a frank attack of Bright's disease occurring in a syphilitic infant such as that reported by Bradley.

Henoch,† who was aware of Bradley's case, stated that he could never establish the connection of infantile renal disease with syphilis, but in the face of Bradley's observation he advised that this should be borne in mind.

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* "On Syphilitic Renal Dropsy," 'Brit. Med. Journ.,' February the 4th, 1871.

† 'Lectures on Children's Diseases,' vol. ii, p. 170.

My first acquaintance with syphilitic nephritis occurred as long ago as 1889, but it was not published until twelve years later.* It arose in a female infant, aged 5 weeks. She first came under observation with desquamation of the palms, the soles of the feet and the abdomen. There were shallow ulcerations on the lips, also in the gluteal fold from the anus upwards, and there were yellow crusts at the root of the nose and on the ears. On the buttocks were a few umbilicated papules. She had chronic "snuffles." Her mother said a rash appeared at the age of two weeks. She was ordered hydrargyrum c̄ creta gr. i thrice daily. A week later puffiness of the hands was noticed. The following week her eyelids were œdematous, white, and waxy looking. The feet and legs and the hands and forearm were much swollen. The urine was albuminous, and hyaline, epithelial and blood casts were found in it. In spite of mercurial treatment, which was persisted in for a month, she died, and post-mortem there was found broncho-pneumonia to account for death, and *the kidneys appeared healthy to the naked eye.* Microscopically, however, the renal changes were decided, and there were both parenchymatous and interstitial alterations. Both were patchy in distribution, and in places the fibro-nuclear infiltration was pronounced (Fig. 27). The fibro-nuclear deposits were most in evidence in the neighbourhood of the glomeruli. The glomeruli were in many instances richly supplied with nuclei; in fact this was the keynote of the glomerular change, but in some few there were other alterations. Thus the following changes were noticed: The Malpighian body was changed, or partially changed into a glassy-looking substance bearing sparsely scattered nuclei; or the substance had a tinge of yellow and a faintly granular aspect; or the combined changes occurred in the same body. In some the faintly yellow exudate was seen in the cavity of the capsule between it and the Malpighian body. The capsule itself was for the most part unaltered, but some capsules were thickened; some had apparently undergone a glassy change, clear or ground glass or a combination of the two. Thickening of the capsule was seen, apart from surrounding fibronuclear infiltration, and the merging of a glassy Malpighian body into a thickened capsule was also observed. In one section there was a tiny gumma consisting of a collection of deeply-staining mononuclear cells, small lymphocytes, and larger

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* 'Syphilis in Children,' p. 56, 1901.

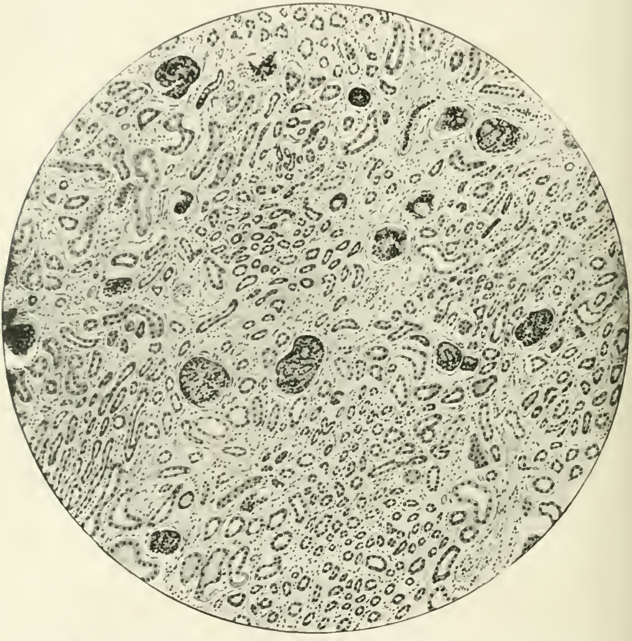

Fig. 27.—Interstitial nephritis in a female infant aged 5 weeks. For description see text. Leitz oc. 1, obj. 4.

cells with round, oval, and indented nuclei margined by a small extravasation of red blood-discs.

This, unlike Bradley's case, unfortunately did not respond to mercurial treatment, and was the first on record to be accompanied by a pathological report.

In another child under my care, aged 6 months, who had an extensive and typical syphilide, there was extreme general dropsy with some free fluid in the peritoneal cavity. Slight swelling of the extremities had been noticed for six weeks. A catheter was passed but the bladder was empty. Unfortunately I lost sight of her.* I have also had † from time to time amongst my out-patients syphilitic infants whose urine suggested catarrhal nephritis. But owing to difficulties incidental to out-patient practice they were imperfectly recorded, and they were also soon lost sight of. Cassell ‡ found albuminuria and formed elements in six out of thirty-one syphilitic infants, but there were no typical examples of Bright's disease among them.

During the last few years our own Society has not been idle, and the 'Reports' contain several records of nephritis occurring in infants. Ashby's § infant, aged 4 weeks, appeared to him to be of the "exact type of scarlatinal nephritis," though he admitted that the suggestion of syphilis which I made at that time was worthy of consideration.

Referring to the cases of nephritis in infants that I have exhibited to the Society, one case was that of a syphilitic infant,‖ aged 5 months. He suffered from dropsy during life and passed albuminous urine containing hyaline, epithelial, and granular casts and red blood-corpuscles. In spite of mercurial treatment (hydrargyrum c̄ creta gr. i, thrice daily) continued for a month he died. Microscopically there was patchy catarrhal nephritis (Fig. 28).

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* 'Syphilis of Children,' p. 57.

† 'Reports of The Society for the Study of Disease in Children,' vol. iii, 1903, p. 292.

‡ *Loc. cit.*

§ "A Case of Nephritis in a Newly-born Infant," 'Reports of The Society for the Study of Disease in Children,' vol. i, p. 129.

‖ "A Case of Syphilitic Nephritis in an Infant, aged 5 months," 'Reports of The Society for the Study of Disease in Children,' 1902, vol. iii, p. 286.

Another example* was that of a dropsical infant, aged 5 weeks, with ascites and with albuminous urine. No microscopical examination of the urine was made. There was neither history nor stigmata of syphilis, and spirochætes, being then unknown, were not searched for. He lived five days. After death there was found bronchopneumonia and collapse of the lungs. There was clear fluid in the abdomen with adhesions between the liver and the abdominal walls. The spleen was twice the natural size. The kidneys showed the following changes: The glomeruli were shrunken and almost hidden by nuclei of proliferated endothelium. The epithelium of the convoluted tubes was mostly broken down into granular detritus, though in some places the cells still lined the walls of the tubes, but their nuclei stained very feebly. In a few tubes there was very active epithelial proliferation, especially in the neighbourhood of the glomeruli. The interstitial tissue also showed much cellular infiltration in the same situation, the combined lesions producing prominent microscopic objects (Fig. 29). A striking feature was the large numbers of lymphocytes and other leucocytes in the veins and capillaries of the liver. Some of the portal canals showed cell infiltrations, lymphocytes, and multinucleated leucocytes extending between the adjacent liver-cells (*vide* Fig. 26, p. 131). There were also changes in the supra-renal capsules (see p. 145).

My last case occurred in a boy, aged 3 months.† Obvious signs of renal disease commenced at 2½ months of age, and were preceded by "snuffles," which occurred somewhat earlier. The urine was albuminous and contained coarse and fine granular casts, hyaline casts with kidney cells therein, kidney epithelial cells, pus corpuscles, and a few red blood-cells. No spirochætes were found. In spite of mercurial treatment the abdomen filled with fluid and he died three weeks later. Post mortem there was extensive pneumococcal peritonitis.

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* "Interstitial Nephritis and Cirrhosis of the Supra-renal Capsules in an Infant, aged 5 weeks," 'Reports of The Society for the Study of Disease in Children,' 1906, vol. vi, p. 258; 'Lancet,' November the 3rd 1906.

† "Syphilitic Nephritis in an Infant," 'Reports of The Society for the Study of Disease in Children,' vol. vii, p. 48; also pp. 286, 287.

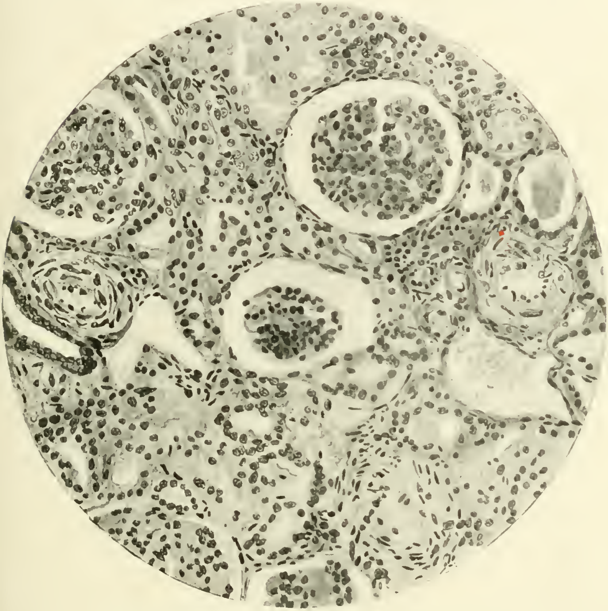

Fig. 28.—Tubular nephritis with prominent interglobular arteries in a syphilitic infant aged 5 months. Leitz oc. 3, obj. 7.

Microscopically the following changes were detected in the kidneys: There was distinct evidence of catarrhal nephritis and the tubules were in places dilated. Scattered areas of interstitial changes were seen from a simple round-celled infiltration, consisting of small lymphocytes and plasma-cells, to a decided fibrosis. Various changes were detected in the Malpighian tufts (Fig. 30). The bulk of them were natural, but others showed varying degrees of hyaline degeneration, in some amounting to complete destruction, and others displayed a shrunken, glassy, slightly nucleated tuft enveloped by a similar substance in the cavity of Bowman's capsule.

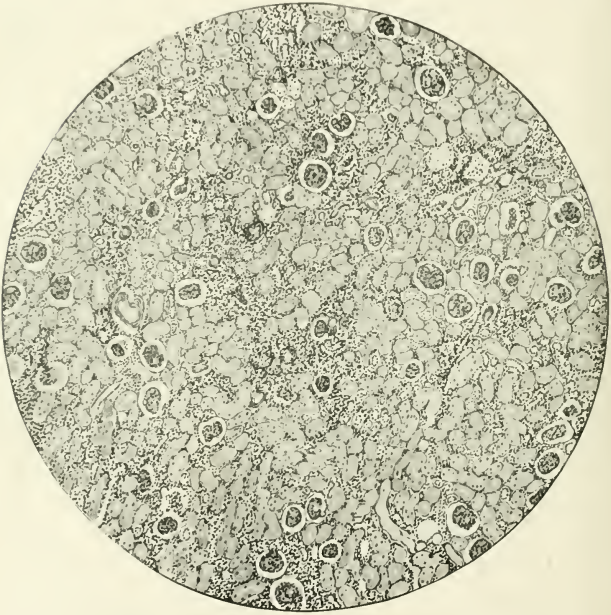

Fig. 29.—Catarrhal and interstitial nephritis from an infant aged 5 weeks (*vide* Fig.). Microscopical section of the liver removed from the same patient. For description see text. Leitz oc. 4, obj. 3.

For the most part the capsules were unaltered, but in some fibro-nuclear thickening was detected. This invaded the tufts, there being in some a general fibrosis. A combination of thickened capsule and hyaline degeneration of the tuft was occasionally noted.

When examining the microscopic sections of the kidneys of these various infants one has been at great pains to determine whether or not the interglobular and afferent or other vessels had altered.

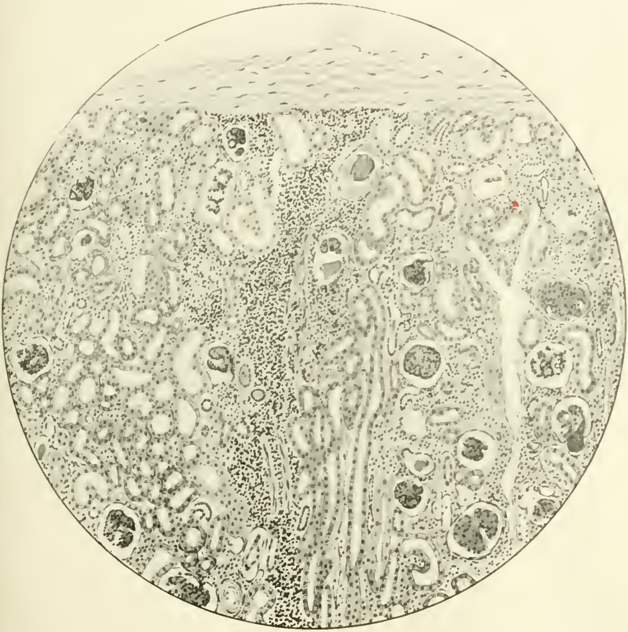

Fig. 30.—Catarrhal and interstitial nephritis in an infant aged 3 months, showing changes in the tubules, glomeruli, and cellular infiltration of the interstitial tissue. For description see text. Leitz oc. 1, obj. 7.

These small arteries were found to be thick walled and generously nucleated, and the impression left on the mind was that they had undergone pathological changes (*vide*

Fig. 28). But on comparing them with normal infantile renal arteries of the same calibre it was seen that the above appearances must be recognised as normal. So, too, the condition of the arterial endothelium. The nuclei of this membrane are naturally prominent. But in some vessels they appeared to have undergone decided proliferation, though here, again, it was felt to be impossible to arrive at any satisfactory decision.

I have elsewhere * drawn attention to the fact that to the *naked eye* the kidneys of infants with nephritis often appear normal, and I think that this is a fact which should be borne in mind.

There can be no longer any room for doubt that the syphilitic virus produces both parenchymatous and interstitial nephritis, in combination or separately.

And it appears that in the infantile stages of the disease—both intra- and extra-uterine—parenchymatous inflammation is not only not unusual, but that this lesion may alone be found at the post-mortem examination. On the other hand, when the typical products of parenchymatous inflammation are found in the urine, it must not be assumed on that account that the stroma is intact.

But as I have elsewhere † pointed out the observations of Fry and Martin ‡ tend to prove that interstitial and combined interstitial and parenchymatous inflammation of the kidneys arise from other than syphilitic toxines. "If that be so another explanation than the syphilitic one is afforded of the origin of the cirrhotic kidney. The possibility also of the seeds of the cirrhotic kidney being sown in infant life must not be overlooked, as also of its origin in intestinal and other toxines." That nephritis, in some cases, commences in intra-uterine life there can be now no question.

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* "Nephritis in Infants," 'Reports of The Society for the Study of Disease in Children,' 1907, vol. vii, pp. 277–288.

† *Loc. cit.*, p. 287.

‡ "Infantile Nephritis," 'Archives of Pediatrics,' January, 1904, vol. xxi, p. 19.

THE SUPRA-RENAL CAPSULES.

The supra-renal capsules are found diseased in some syphilitic fœtuses—about one in eight. These have been noticed larger than natural by Virchow, Hecker, and others.* In some cases the increase in size has been due to disseminated nuclei and young connective tissue, as in the liver, and the new growth appears to be very liable to fatty degeneration, so that the organ may be found completely transformed into a mass of oil-drops and granular detritus.

In one of the cases that I brought before the Society † the supra-renal capsules were greatly enlarged, being nearly the size of the kidneys (see p. 140). On cutting into them they each showed yellowish-brown discoloration, apparently the result of old hæmorrhages, lying in the midst of an opaque white substance. On microscopic section, taken at the junction of the opaque white medulla and the healthy cortex, the opaque white substance was seen to be fibro-cellular tissue, very rich in polymorphous nuclei, containing hyaline masses.

These hyaline masses were of irregular oval shape, occupied about the fourth of the field of a high power, and stained fairly well; they contained a few nuclei of various shapes, and they sent processes into the surrounding fibrous tissue.

THE HEART AND GREAT BLOOD-VESSELS.

The heart, like other organs of the body, does not escape attack. Myocarditis has been found in syphilitic fœtuses ‡ and it has been demonstrated in the heart of an infant, aged 6 weeks, by Hektoen. § In a case observed by Kantzow[*] adjoining an interstitial myocarditis there was seen a muscular hypertrophy (myoma) which Virchow attributed to the neighbouring irritation, similar to that which produces bone formation in the vicinity of a gumma of the periosteum. In Coupland's case[†] the heart muscle was firm and resisting, and of a uniform pale pinkish-yellow tinge; the walls were thick, and they cut with a creaking noise. There was an extensive infiltration of small round cells, most abundant round the small arteries; the muscular fibres retained their normal striated appearance. Rosen[‡] records a congenital gumma of the left heart and we have seen here an example of gumma of the heart in an infant, aged 3 months, which was shown by Porter Parkinson.[§] Forster[||] describes syphilitic endocarditis in a six months old infant.

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* Lancereaux, "Des Affections Viscerales de la Syphilis Héreditaire," *loc. cit.*

† "Interstitial Nephritis and Cirrhosis of the Supra-renal Capsules in an Infant aged 5 weeks," 'Lancet,' November the 3rd, 1906.

‡ "Syphilitic Fibrous Myocarditis in an Infant born dead," Wagner, 'Das Syphilom Archiv der Heilkunde,' vol. vii, p. 527.

§ 'Syphilis of Children,' p. 57.

I have elsewhere called attention to congenital syphilis as a cause of congenital heart disease, and increasing experience makes me feel more than ever that therein lies an explanation for some cases of fœtal endocarditis and carditis. I have seen a case of coarctation of the aorta in a boy, aged 5 years, which was probably of syphilitic origin, *vide* Case 1 in the appendix. Le Boutellier,[¶] who has collected a number of cases of extra-cranial aneurysm occurring in the young, has referred to a fœtus of 8 months with an aneurysm of the abdominal aorta. He has collected seven cases of thoracic aneurysm in children under ten years, the youngest of whom was two years. In many instances the aneurysm was ascribed to syphilitic arteritis. Robert Wilson and Alexander Marey, jun.,[**] reported in 1907 the case of a child, aged 4 years, who died of a ruptured aortic aneurysm of the ascending and transverse aorta. It had stenosis of the aortic orifice, which was almost occluded, along with patches of atheroma. An aortic bruit dated from infancy. Both the valvular lesion and the aneurysm were considered to be of congenital origin and also due to congenital syphilis. The radial arteries were thickened, and there was general adenitis.

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[*] 'Archiv der Path., Anat., und Physiol.,' vol. xxxv, p. 211, 1866.

[†] *Loc. cit.*

[‡] 'Behrend's Syphilidologie,' vol. iii, p. 249, 1860.

[§] 'Reports of The Society for the Study of Disease in Children,' vol. i, pp. 141–142.

[||] 'Waerzburg. Med. Zeitschr.,' vol. iv, p. 7, 1863.

[¶] 'American Journal of Medical Science,' 1906.

[**] 'Trans. Col. of Phys., Philadelphia,' third series, vol. xxix.

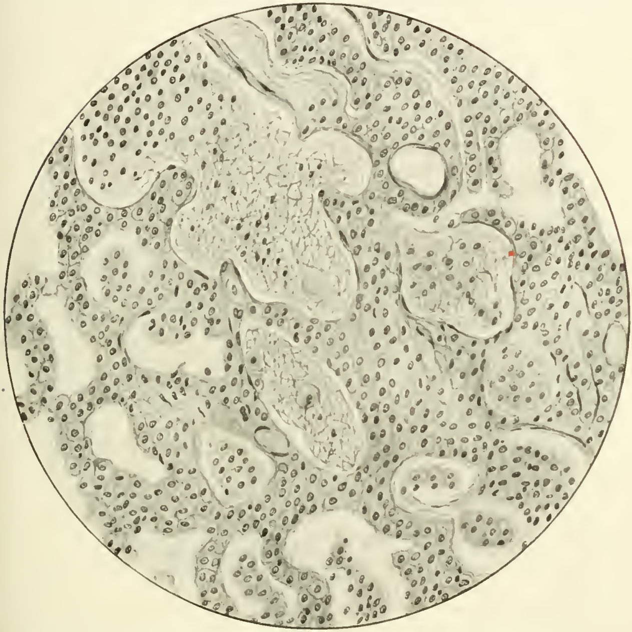

FIG. 31.—Syphilitic lung showing thickened alveolar walls from proliferated endothelium. Alveoli plugged with fibrin, and showing leucocytes and catarrhal cells. Leitz oc. 3, obj. 7.

SYPHILIS OF THE MOUTH, THROAT, LARYNX, AND LUNGS IN INFANTS.

Syphilitic affections of the mouth, throat, larynx, and lungs are not common in infants, though various lesions of the fœtal organs have been recorded.

Such complaints usually occur as later manifestations, but even deep ulceration of the palate, pharynx, and nasopharynx have arisen in early infancy.

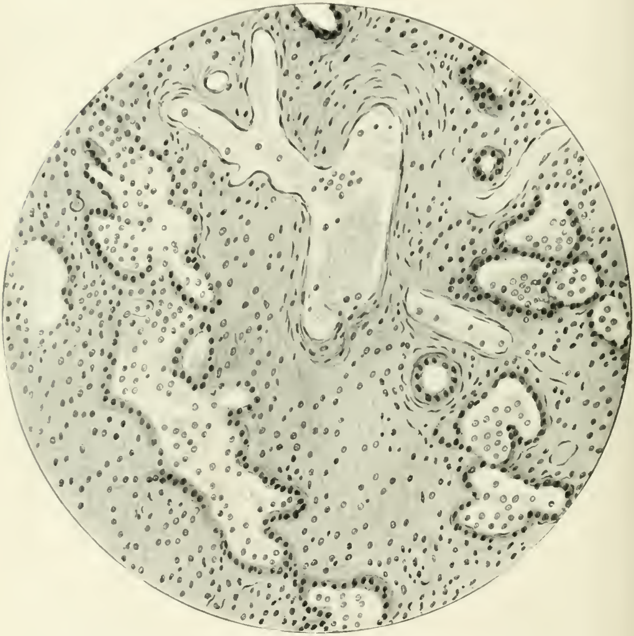

Fig. 32.—Syphilitic lung showing dilated vascular channels, fibroid induration, and lining of the alveolar remnants with a cubical epithelium. Leitz oc. 3, obj. 7.

In an infant under my care, aged 4 months, the aryepiglottic folds were swollen, and an ulcer perforating the thyro-hyoid membrane led to a thin-walled cavity underneath the muscles. There was a dorsal ulcer at the posterior third of the tongue. The periosteum above the left knee was thickened.* During life the cry was aphonic and the breathing stridulous.

In a child, aged 13 months, I found ulceration of the larynx and white hepatisation (first described by Devergie in 1831 †) of the lung, *vide* Appendix, Case 3.

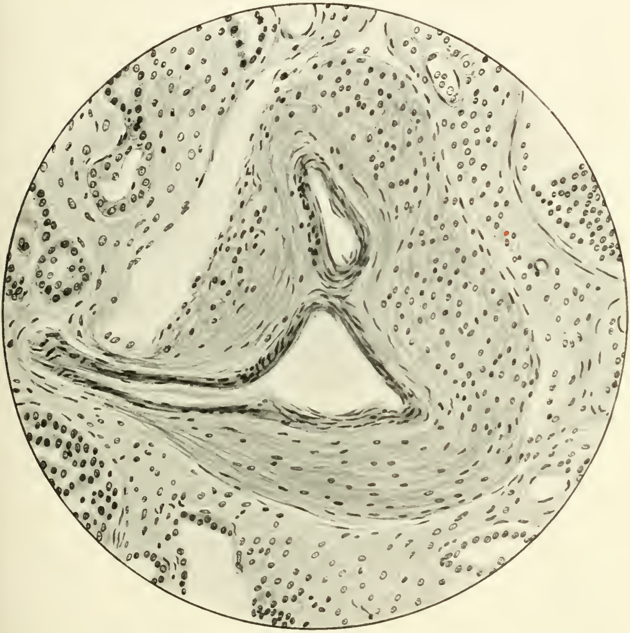

Fig. 33.—Syphilitic lung showing thickening of the external coat of an artery, fibroid thickening of the alveolar walls, with disappearance of the alveoli. Lymph space above and to left of artery. Leitz oc. 3, obj. 7.

In a syphilitic child, aged 1 month, there was a fleshy consolidation (splenisation) of the lung of a fibroid nature. The microscopical appearances were as follows:

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* 'Syphilis of Children,' p. 43, par. 2.

† 'Annales d'Hygiéne et de Medécine Legales,' April, 1831.

The alveolar septa show the following changes: The alterations in them range from a condition of dilated capillaries in thickened alveolar walls which appear to consist solely of proliferated endothelium (*vide* Fig. 31) to a state where the thickening is definitely fibrillar with polymorphous nuclei and enlarged endothelial vascular channels (*vide* Figs. 32 and 33). Some of the capillaries in these walls show extravasation of leucocytes.

The alveoli are stuffed with cells so that it is often hard to separate the wall from its contents. These cells are mostly of the ordinary catarrhal variety with single well-staining nuclei of good protoplasmic envelope. But there are also cells twice or thrice that size of granular or pigmented aspect with poorly staining single, sometimes double, nuclei. Along with some of these cellular contents is granular detritus. The first mentioned have nuclei somewhat larger than a small lymphocyte, and in regard to protoplasm they greatly exceed the amount allotted to that body. In parts attenuated alveoli have put on an appearance as of a lining of cubical epithelium apart from the broncho-pneumonia (Fig. 32).

Some of the alveoli are plugged with fibrin which is associated with catarrhal cells, and some cells are so poorly supplied with a protoplasmic envelope that they would do well for small lymphocytes (*vide* Fig. 32).

Blood channels (*vide* Fig. 32) possessing only an endothelial lining are thickened by surrounding fibrous deposit. Many greatly enlarged capillaries are seen ramifying in the thickened alveolar walls. Some of these blood channels (veins), seen to commence in definite capillaries, display fibrin clots with enmeshed small lymphocytes, and in some small lymphocytes are found blocking these latter. Such small clots can also be seen in the vasa vasorum within the arterial coats and in the blood-vessels within the bronchial walls. Large lymphocytes are only seen in them occasionally.

In one or two arteries in the sections there was pronounced thickening of the external coat (*vide* Fig. 33).

Attention has been drawn to plugging of the capillaries and small veins with lymphocytes and fibrin. This is a feature which I have also observed in the broncho-pneumonia of congenital syphilitics.

The small yellow gummata which are occasionally found post mortem in infants' lungs have much the same microscopical appearances as gummata in other organs. In their central parts they are apt to be definitely fibrous, and at their periphery they are chiefly composed of small round cells which invade the alveolar septa. In their growth the alveoli are destroyed, and all stages of their obliteration can be seen in the microscopic fields, where they occur as irregular spaces bounded by cells. The bronchial tubes are either stuffed with catarrhal products or have shed their linings, and their walls are infiltrated with small lymphocytes.

I have also met with several examples of extensive consolidation of the lungs from the apices downwards in syphilitic infants which has persisted for long periods. The youngest was six months old, and consolidation was known to be present from March the 31st to November the 24th, when she ceased attendance. She died from being scalded the following January. Whether they were examples of delayed resolution in pneumonia or of syphilitic pneumonia it is, I think, impossible to say. Syphilis has to be thought of in such cases. But the further removed the case is from the syphilitic age the greater are the chances of the consolidation being tuberculous. Even in the post-mortem room sometimes consolidation may look simple when it is in reality tuberculous. A syphilitic infant a year old, with consolidation of the upper half of one lung, suffered from some fever and loss of flesh. Anti-syphilitic treatment had no effect, and he died some few weeks later. The upper half of the lung was consolidated and of a red colour. Its bronchial tubes were thickened, dilated, and contained muco-pus. Radiating from the bronchial tubes were scars of different sizes passing in various directions and intersecting the solid areas in varying degrees. The alveoli were stuffed with fibrin, with fibrin and catarrhal cells, and with catarrhal cells only. The alveolar walls showed considerable endothelial proliferation. The thickened endothelium was characterised by increased vascularity and by a fibrillar or reticulate stroma. Many alveoli were collapsed. In the consolidated areas were quite a number of giant cells apparently formed from the endothelium. In one small area there were signs of commencing caseation. The septa were thickened. The bronchi showed infiltration of their walls with round cells and some had shed their epithelium. On the other hand some cases of cirrhosis of the lung in young children may well be syphilitic, and modern methods of examination may prove them to be so. The spirochæte has been detected in the sputum of those suffering from syphilitic pneumonia, and the discovery of this organism will enable us in the future to more readily recognise this condition of the lung.

The child Isabel H—, aged 5 years, who died of exploratory puncture of the chest, and an account of which I published in 'The American Journal of the Medical Sciences,'* had an indurated, shrunken, and scarred lung, with dilated and thickened bronchial tubes, and which, microscopically, could not be differentiated from syphilitic pneumonia. It was not tuberculous, but there was no history of syphilis.

Syphilis of the Testicles.

Henoch, Déspres, R. W. Taylor, Obédenare, North, Bryant, Parrot, Lewin, Hutinel, Fournier, and Seringe have recorded cases and done original work on the testicles of congenital syphilitics.

In September, 1892, I published some observations on hereditary syphilis of the testicles in the 'Practitioner,' and I arrived at the following conclusions:

Syphilitic inflammation may take the form of the gummatous or the cirrhotic (Figs. 34 and 35). I have seen both varieties. All parts of the organ may be attacked, and even the cord and the vas may feel thickened. Hydrocele is not an uncommon association.

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* October, 1893, vol. cvi, No. 4, pp. 422–423.

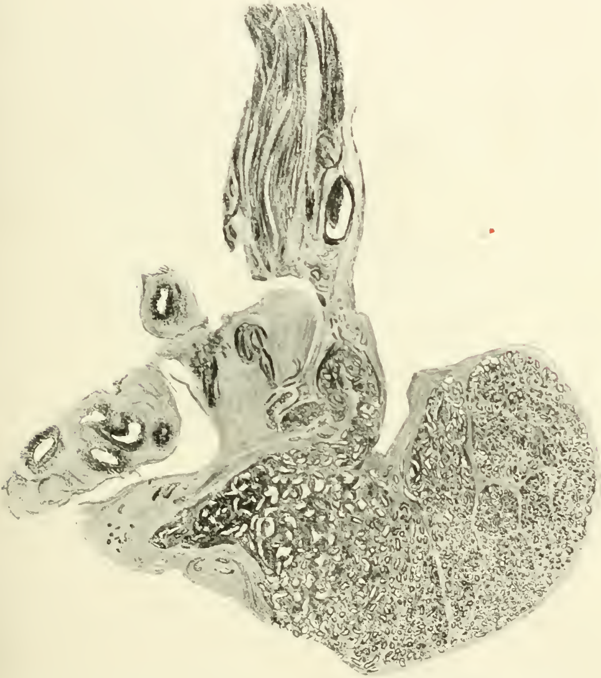

Fig. 34.—Syphilitic testicle removed from an infant, aged 6 weeks. × 10, and showing cirrhosis of the body and the epididymis. For an account of the case see footnote to Fig. 35.

The infantile testicle in health varies in size between a lemon-pip and a hazel-nut, and the normal tension of the globe is, in the language of ophthalmology, about + 2.

The left testicle is usually the larger of the two. When the organ is enlarged the recognition that there is something amiss is not difficult, but when there is no enlargement, and enlargement is not a necessary accompaniment of disease, and when reliance has to be placed entirely on testicular tension, then the chance of arriving at a wrong conclusion is by no means small, because it is very difficult to secure and test for fluctuation so small and so elusive a body as the infantile testicle. But if there be cirrhosis

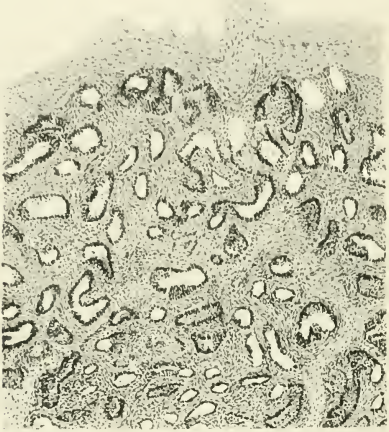

Fig. 35.—Microscopic section × 50 of the testicle of a syphilitic infant, aged 6 weeks, showing cirrhosis of the organ. In the normal testicle the seminal tubules would be close together, and the amount of areolar tissue but trifling. The tunica albuginea is thickened. The infant had "snuffles" and multiple syphilitic epiphysitis of a week's duration. The hands and feet were freely desquamating. He had a confluent eruption on the buttocks and the calves, surrounded by outlying flat-topped papules the circumference of a split-pea, a confluent syphilide round the mouth reaching to the neck, and shallow ulcers on the lips. The axillæ displayed a scaly eruption. There were hydroceles on both sides, the epididymes felt enlarged, and were certainly hard. The cords also seemed enlarged. The hydroceles cleared under mercurial treatment, and the testicles then felt hard, but were not enlarged. He died after a month's treatment.

of the organ, and if there be no associated hydrocele to hamper the examination, in place of the normal tension of the globe there may be felt a hardness which can be compared with that of scirrhus.

On the other hand, the organ may enlarge to the size of a filbert, a chestnut, a French plum, or even an egg,* and in that case the choice would lie between syphilis, tubercle, and a new growth, all being possible.

So far neither the seminal vesicles nor the prostate have been found enlarged in syphilis, the only present clinical distinction between the tuberculous and the syphilitic testicle. Calmette's tuberculin ophthalmic reaction should here prove useful, seeing that a tuberculous testicle has been found in a typically syphilitic infant (Stoeber). But a diseased testicle in the syphilitic age of life is more likely to be syphilitic than tuberculous.

Certain cases of infantilism must be ascribed to double syphilitic orchitis. It also accounts for mal-development of the organ. In regard to this latter it will be remembered by some of the members of this Society that Mr. Anglin Whitelocke showed an ovary-looking organ † which he had removed from the inguinal hernial sac of a female child, and which was thought by the members present to be a badly developed testicle. A microscopic drawing of this was shown, and the similarity between it and that of a syphilitic cirrhotic testicle (*vide* Fig. 35) was most striking.

SYPHILIS OF THE OVARIES AND INTERNAL GENITALIA.

I have no records of sclerosis of the ovaries, but the *Treponema pallidum* has been demonstrated there, and these organs have been found diseased in the syphilitic fœtus. Cases of syphilitic infantilism occur also in females, and some examples of this condition I have no doubt are to be explained by syphilitic atrophy of these organs.

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* 'Syphilis in Children,' pp. 59–63.

† "Notes on a Case of Double Inguinal Hernia in a Female Child, each Sac containing an Ovary-like Organ," 'Reports of The Society for the Study of Disease in Children,' vol. ii, pp. 191–195.

But I have seen one case of ovarian disease which was possibly syphilitic in origin and gummatous in nature. In 1903 I showed to the Society,* at the discussion on tuberculous peritonitis, as an illustration of the value of bimanual examination in abdominal disease, a drawing of an ovarian tumour with a dilated Fallopian tube which had been diagnosed by me by rectal bimanual examination during life. The infant, who had not been healthy from birth, was twenty-two months old. In the right inguinal region a tumour was found which did not feel fixed even through the abdominal wall. By rectal examination the uterus was normal. Attached to its upper part by a short, cord-like attachment was the tumour, and on the posterior surface of this tumour was a convoluted tube. The tumour was freely movable and could be carried over to the mid-line of the body, though not beyond it. Fluctuation was detected. The ovary could not be felt. On the opposite side it was healthy. The child had Parrot's nodes, the free edge of the spleen projected four fingers' breadth in

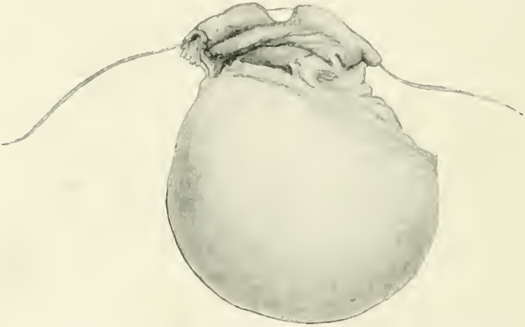

FIG. 36.—Drawing of the ovarian tumour and dilated Fallopian tube from a child aged 22 months, as it appeared after removal.

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* "Tuberculous Peritonitis, Symptoms and Diagnosis," 'Reports of The Society for the Study of Disease in Children,' vol. iii, pp. 90–91.

the front of the costal margin, and the liver three fingers' below it. The tumour was about the size of a double walnut ($1\frac{3}{4}$ in. by $1\frac{5}{8}$ in.) and contained foul-smelling pus; it was slightly adherent to the rectum, the cæcum, and the bladder. The conclusion arrived at was that it was an ovarian cyst burrowing down between the layers of the broad ligament. The Fallopian tube, which was enlarged and curly, could be felt behind and above it. It was not tuberculous.

SYPHILIS OF THE NERVOUS SYSTEM IN INFANTS.

Of disease of the nervous system in infants I have not met with many examples. Syphilitic meningitis, spots of yellow softening of the brain, endarteritis, and gummata on the cranial nerves, perhaps associated with choroiditis, may be met with occasionally, but, judging from the paucity of the literature they are of very rare occurrence. Cases illustrating these conditions in infants have been published by Barlow, Chiari, and Henoch,* Sutherland and Thompson Walker,† and others.

I have seen several cases of hydrocephalus in syphilitic infants; one recovered under mercurial treatment, the others I lost sight of. Fruhinsholz ‡ gives an account of the post-mortem examinations of two cases of syphilitic hydrocephalus.

In a syphilitic infant, aged 4 months, with the clinical signs of hydrocephalus; the effusion of fluid was found between a thin, yellowish-white, friable arachnoid and the underlying pia mater. The pia mater displayed diminutive, yellowish, sharply-defined tracks of thickening and hardening, which microscopically displayed round-celled infiltration, especially abundant at its outer part and in the neighbourhood of the vessels.

In another infant, aged 6 months, with the symptoms during life of basic meningitis, in whom after death there was found internal hydrocephalus, the following were detected: Diminutive, scattered, localised, whitish infiltrations of the meninges. Also partial sclerosis of the choroid plexuses, the vascular walls showing fibrous changes, together with occlusion of their channels, with similar formation or with embryonic cells. The ventricular surfaces of the optic thalamus and corpus striatum were covered with thickened membrane. In the meshes of this fibro-nuclear tissue were numerous collections of round cells together with a plentiful vascular supply. Surrounding the blood-vessels in the superficial part of the membrane were several layers of round cells.

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* 'Syphilis in Children,' pp. 77–80.

† *Loc. cit.*, pp. 134–146.

‡ *Loc. cit.*, pp. 44–48.

Fruhinsholz also describes in syphilitic infants localised cloudiness and thickening of the meninges and gummata the size of miliary tubercles on the cerebral vessels. These were accidentally discovered and would have been easily passed on a cursory examination.

He also found in the frontal lobe of the brain of an infant, aged 15 days, a depressed area with thickened, adherent, yellowish-white meninges covering cerebral softening of the appearance of yellow pap and of the size of an olive.

I have had under my care a syphilitic girl of five and a half months with extensive encephalitis, for a full record of which *vide* Appendix, Case 3.

APPENDIX.

1. A CASE OF COARCTATION OF THE AORTA.

Percy S. W—, aged 5 years, was brought by his mother to the Evelina Hospital on June the 10th, 1895. The labour was a difficult one, and he was born with "snuffles." Six weeks before his birth his mother was laid up with "influenza and pleurisy." There had been five children of the marriage, one of whom had died from "inflammation of the brain" at three years. All the children suffered from "snuffles" at birth, and "some of them had rashes." There had not been any miscarriages or still-born children. There was a history of rheumatic fever in both parents, and phthisis on his mother's side. He was a badly-nourished, ill-developed child, with a pallid face, oral cyanosis, and loud and laboured breathing. On stripping him for examination what immediately attracted my attention was the enlargement and tortuosity of the arteries about the scapular and abdominal regions. In the armpits the subscapular and long thoracic arteries were thick, tortuous, and pulsating. The pulsation could be distinctly seen, and the arteries felt very hard. A thick pulsating vessel could be felt crossing the subclavian triangle on both sides. This appeared to be the transverse cervical artery. The superficial cervical artery was enlarged on the left side In the eighth and ninth interspaces behind on the left side enlarged tortuous and pulsating vessels could be seen and felt running from within outwards, and other vessels were noticed crossing the posterior border of the scapula. Similar vessels were seen on the right side, but they were not so well marked or so much enlarged. The axillary arteries felt enlarged on both sides, and hard, and they were decidedly tortuous. The brachial arteries felt like whipcord; pulsation was well marked in them and they were slightly tortuous. The radial arteries did not appear to be thickened. A feeble pulse at the wrist was detected in both the radial and ulnar arteries. The superior epigastric and superficial epigastric arteries could be seen pulsating and felt beneath the skin. The former was decidedly enlarged and tortuous, more so on the left side. The femoral arteries were small, felt like rigid cords, and very feeble pulsation could be detected in them. Feeble pulsation was felt in the anterior and posterior tibial arteries. The abdominal aorta could be felt feebly pulsating through the abdominal wall. *Per rectum* two cord-like structures were distinguished along the brim of the pelvis, one on either side; no pulsation could be felt in these. The deep epigastric arteries could be grasped bimanually and were found to be enlarged and tortuous. A number of other large vessels could also be felt in the abdominal wall. A collection of enlarged and tortuous vessels was detected in the pelvis, especially in the neighbourhood of the sacro-sciatic notch, feeling like a collection of worms on either side.

Running down the front of the sacrum a large and thick vessel was found, and coursing along the posterior wall of the rectum was another enlarged and tortuous vessel.

One of the superficial arteries of the abdomen was removed for microscopical examination. During its removal the vessel did not pulsate like an artery, the flow of blood being venous both in colour and appearance. When a microscopical examination of the vessel was made no fault could be found with it—it had all the appearances of a perfectly healthy artery. The thickening of the vessels was, therefore, due to well nigh empty and contracted arteries.

Heart.—The cardiac impulse was somewhat diffused, but best seen and felt in the fifth interspace in the nipple line and of a heaving character. Slight epigastric impulse was also detected. The area of dulness did not extend to the right and commenced at the third left costal cartilage. The sounds were natural at the apex, but basic systolic bruits were audible on both sides. The murmur was heard rather better on the right side than on the left, and conducted better to the right clavicle than to the left. The bruit was also heard over the right chest behind and on the right axilla and detected over the spine of the left scapula. It was not at all striking.

Lungs normal.

Liver.—The free edge could be felt three fingers' breadth below the costal margin in the nipple line.

Spleen.—This projected two fingers' breadth in front of the costal margin.

Abdomen natural.

Urine normal.

Osseous system.—The skull appeared to be thickened. Bossing was felt on the parietal bones near the coronal suture. The occipital bones appeared thickened, and there was some doubtful thickening of the temporal bones.

Skin.—At the left angle of the mouth there were some linear scars. In the right buttock near the anus were two very small circular scars. A small scar was found just below the left patella. The fingers and toes showed a slight tendency to clubbing.

Naso-pharynx.—The boy had adenoids and naso-pharyngeal catarrh and cervical adenitis on both sides of some extent.

Fundus oculi.—Nothing abnormal was detected. While under observation he steadily emaciated and suffered from fever. Occasional puffiness of the face and blueness of the lips were seen; the breathing became loud and laboured at times with occasional coughing. At times he was drowsy by day and restless by night. I made a further examination of the fundus oculi on June the 19th and found tubercles of the choroid of common type and early optic papillitis. The parents, understanding his case to be hopeless, removed him, and the diagnosis could not be verified. But I believe the boy was suffering from a congenitally stenosed or occluded aorta at the point of junction of the ductus arteriosus and of syphilitic origin.

2. A CASE OF CONGENITAL SYPHILITIC ULCERATION OF THE LARYNX AND INTERSTITIAL PNEUMONIA.

Mary E. S—, aged 13 months, was brought to the out-patient department at the Evelina Hospital on October the 29th, 1894, by her mother, who stated that she had "bronchitis."

The mother had been delivered of ten children, but five of these were still-born; she had never had any miscarriages. All of the children had thrush, "which passed through them, but did not extend below the buttocks." None of her children suffered from "snuffles," but the subject of these notes had considerable muco-purulent discharge from the nose, and the mother owned that the child had been troubled with this since she was three months of age, at which time "a rash first broke out round her mouth and then passed to her bottom, but did not extend as low as her knees." She was taken to Guy's Hospital at this time, and was given grey powders.

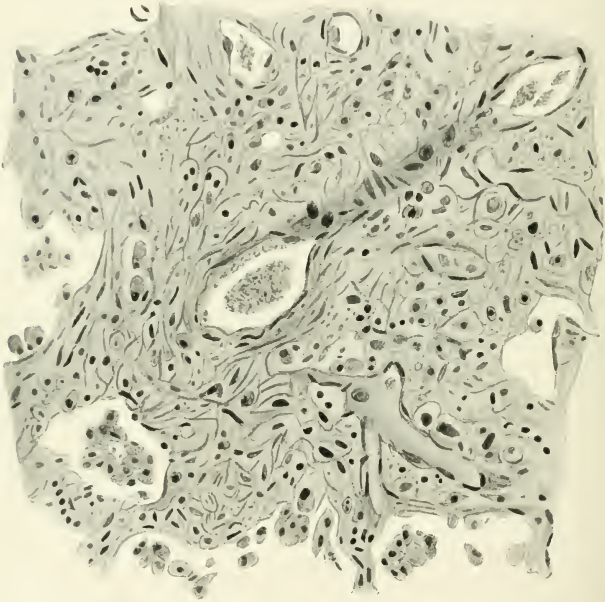

FIG. 37.—Syphilitic lung showing dilated capillaries, fibro-nuclear thickening of alveolar walls, and destruction of alveoli. Catarrhal cells in alveolar remnants. Leitz oc. 3 obj. 7.

On examination.—The head was small, measuring 15½ in.

in circumference, and the anterior fontanelle was widely open. Her mother said she was "very knowing, always laughing and talking in her little way at home." Whilst I was examining her she had an attack of laryngeal spasm. The crowing was not loud and noisy, as with healthy vocal cords, but was dysphonic. Her mother then volunteered the statement that "ever since she had first been taken bad she had crowing on and off." The mouth was extensively scarred at the angles, and the sides of the cheeks and the chin were invaded.

The right upper lobe of the lung was dull to percussion, but little information was obtained from auscultation of the lungs, as a whistling sound of laryngeal origin was audible all over the chest.

There was an eruption on the buttocks and vulva, consisting of papules, varying in size from a pin's head to a small split-pea. These had an excoriated look, and were red in colour. On the backs of the upper part of the thighs were stains, varying from a pin's head to a pin puncture, and of a dusky hue.

She was treated for syphilis. A few days after her first appearance at the hospital she died somewhat suddenly.

Autopsy.—Larynx : The epiglottis and ary-epiglottic folds were much swollen.

There was extensive ulceration of both vocal cords, the larger being on the left side, the edges of the ulcers being ragged and their bases rather deep. Both ventricular bands were also ulcerated, the ulceration extending to the ary-epiglottic folds; this was much more marked on the right side than on the left. The under surface of the epiglottis had a mossy appearance, and when the organ was opened out for the purpose of sketching, deep fissures came in view.

Lungs : Somewhat recent adhesions were found on the right side. The whole of the upper lobe on the right side had a greyish, slightly granular appearance, and was quite solid. Neither air nor fluid could be squeezed from its cut surface, and it could not be readily broken up by the thumb-nail. Some delicate strands of fibrous tissue were seen in it. The other lobes were perfectly healthy.

A microscopical examination of the consolidated lung revealed the following changes:

The alveolar septa show definite alterations in structure, ranging from a condition of dilated capillaries with but few nuclei to a state where the walls are thickened by fibro-nuclear tissue. This tissue occurs either as very delicate fibrillæ with round or oval nuclei, or as a pronounced and fibrous-looking structure with nuclei of all shapes, round, oval, fusiform, and pyriform as in areolar tissue (Fig. 37).

In the alveoli are seen granular detritus with ordinary simple nucleated well-stained catarrhal cells, or with these and with cells of twice, or thrice that size with single or double nuclei. Mostly there is no detritus, and in those situations where the thickening of the alveolar walls is pronounced and the catarrhal cells are arranged in clumps they affect a carcinomatous aspect; there are no fibrin plugs to be seen. A prominent microscopic feature is the number of vascular channels which possess but an endothelial lining, are enveloped by a mass of fibro-nucleated tissue, and take on a circular form. These new vascular channels are very numerous.

The bronchi have shed their epithelial contents, and in some catarrhal cells, small lymphocytes, and granular detritus are visible in their interiors, and their walls are plentifully infiltrated with round cells, being prominent microscopic objects under a low power. The infiltration consists of small lymphocytes mostly, and a few plasma-cells. The matrix in which they lie is either structureless or displays faint fibrillation, or is unmistakably retiform. Numerous capillaries course through these little neoplasms, and all stages of tissue formation can be seen in progress in them up to a decided fibrosis with polymorphous nuclei.

In several bronchi are large polypoid outgrowths of fibro-nuclear tissue into the lumen of the tubes.

Around some bronchi and elsewhere in the section are circular clumps of round cells—small gummata. These round cells are mostly small lymphocytes with a few plasma-cells. In places a retiform tissue can be defined, but elsewhere this is not obvious. But what is obvious in some of them is a commencing fibrosis—the fibrillæ are pronounced, there is increased vascularity and polymorphous nucleation.

The vascular channels contain red blood discs, but there is no clotting of the blood and no leucocytosis. Comparing the specimens with healthy lung tissue in infants I cannot persuade myself that the arteries or veins are altered. Thickened external walls they have, the arteries especially, but such is the appearance in normal lungs. When there is thickening of the external vascular coat this appears to be due to encroachment of fibrosis rather than to changes occurring within the vascular walls.

3. A CASE OF SYPHILITIC CORTICAL SCLEROSIS (ENCEPHALITIS) IN AN INFANT AGED 5½ MONTHS.

Ethel S—, aged 5½ months, came under my care at the Evelina Hospital on February the 13th, 1893. Her mother had had one other child, which was born dead, and "her hair came off in large quantities afterwards." The infant was perfectly healthy at birth with the exception of its "eyes, which appeared to be crossed." At one month it was unable to suck, and had "snuffles." Then convulsions began one morning by a sudden movement of the left arm. The mother was struck in the breast. Then the movements spread to the right arm and then to the legs. The head became retracted, and it remained so from that time. At two and a half months she "moved her eyes in a peculiar manner, and she was pronounced to be blind by a doctor." When she came under my notice she was a fairly nourished infant. She had "snuffles," and a macular eruption on the buttocks. The face was flushed, the respirations rapid, 70 to the minute, and grunting. The bridge of her nose was depressed and widened. Her eyes were moved in every direction, but usually were aimed slightly upwards and to the left side. The head was markedly retracted, and all the limbs were stiff. The fingers were flexed into the palms, and the thumbs flexed and adducted. The arms were generally flexed and adducted, but they were extended at times. The toes were flexed and adducted, the legs flexed on the thighs, and the thighs on the abdomen.

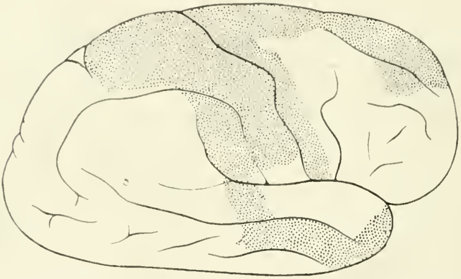

FIG. 38.—Brain of Ethel S—, aged 5½ months. Right hemisphere, showing sclerosed areas. From a sketch made by the author at the autopsy. For description see text.

Fundi oculorum.—Patches of choroidal atrophy were seen in both fundi.

There was some slight broncho-pneumonia, and the temperature was 99·2° F. There were no physical signs otherwise. She died on the 16th.

Post-mortem, February the 17th.—Rigidities of the arms and legs were marked, and there was green discoloration of the abdomen. The lungs showed patchy broncho-pneumonia, and the eye-backs patches of choroidal atrophy. The other organs were normal.

The brain showed areas of atrophy of both hemispheres on their external and internal aspects. On removing the skull cap the place of the atrophied areas was seen to be filled up by cystic formation. The cysts were loculated, and the loculi contained either clear or slightly opaque fluid, their walls as if sanded with lymph and with no trace of blood or crystals. Parts, however, retained the shapes of the convolutions, and these, though small, were hard and dense on section. The sclerosis of the right hemisphere (Fig. 38) on its outer surface involved the following convolutions—the ascending parietal and the parietal lobule; that portion of the ascending frontal (about half its width) in proximity to the fissure of Rolando, starting from the fissure of Sylvius; the superior frontal and a portion of the middle frontal; a portion of the superior temporo-sphenoidal at the commencement of the fissure of Sylvius, and the front half of the middle temporo-sphenoidal gyrus running into the tip of the temporo-sphenoidal lobe. On its inner aspect the sclerosis invaded the following gyri, viz. part of the callosal, part of the marginal, and part of the quadrilateral. It reached from the corpus callosum below to the vertex above, where it joined the other affected areas.

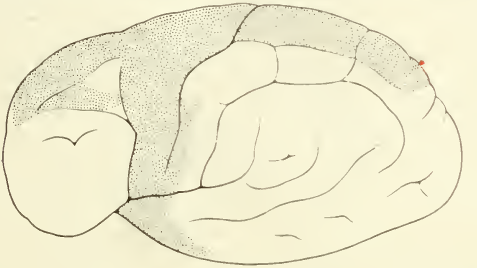

FIG. 39.—Brain of Ethel S—, aged 5½ months. Left hemisphere, showing sclerosed areas. From a sketch made by the author at the autopsy. For description see text.

The affected gyri of the left hemisphere (Fig. 39) on the outer aspect were the ascending frontal, the parietal lobule and the first annectant, a narrow strip of the ascending parietal adjoining the fissure of Rolando from the fissure of Sylvius upwards, the superior and part of the middle frontal, and a small area of the superior temporo-sphenoidal adjoining the fissure of Sylvius and slightly invading the middle temporo-sphenoidal gyrus.

The inner surface of this hemisphere showed sclerosis of part of the callosal and marginal gyri stretching from the corpus callosum and reaching to the affected areas on the vertex.

The right hemisphere was smaller than the left.

Cross sections of the brain showed the sclerosed areas to be sharply defined (*vide* Fig. 40, Nos. 1—5).

The large arteries were unaltered. No naked-eye changes were found in the spinal cord. There was slight patchy broncho-pneumonia and the other organs were normal.

Histological Report by Dr. Ernest Jones.

The following report is based on seven sections of the brain of Dr. George Carpenter's case. Three were taken from the cortex and four from the white matter. As the sections were made and stained (by hæmotoxylin and eosin) some fifteen years ago, it was impossible to study the finer changes, particularly those relating to the glia and nerve cells, which are so important in connection with congenital lues. The following facts however were clearly observable.

(A) *Pia mater.*—In general this was not appreciably thickened or adherent, but in places there was a considerable exudation between the pia and cortex. There was a diffuse cellular infiltration present, particularly of small mononuclear cells. This infiltration bore a distinct relation to the vessels. The vessels themselves were not greatly altered. There was no endothelial proliferation, and the most striking change was a small-celled infiltration of the adventitia, where a few plasma-cells were also to be seen.

(B) *Cortex.*—So far as the nerve-cells are concerned the

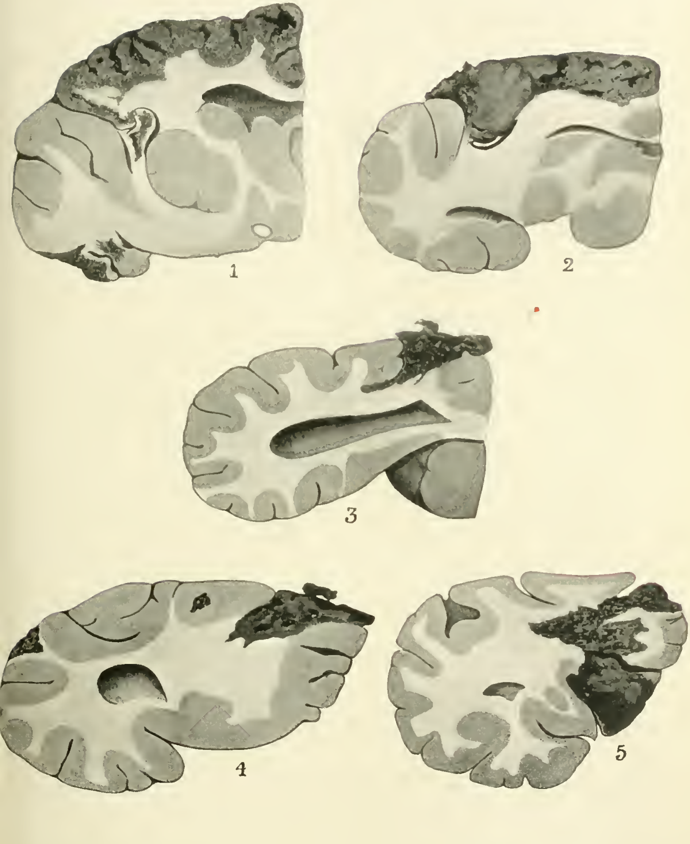

FIG. 40.—Brain of Ethel S—, aged 5½ months. Transverse sections from before backwards, showing the extent of the sclerosed areas. From sketches made at the autopsy by the author. For description see text.

only facts that can be stated in regard to them are: (1) The differentiation of the different layers showed a decided retardation in relation to the age of the child; (2) the cells stained badly; (3) no obvious deformity or diminution in number could be made out.

There was a decided increase of the glia tissue, though the number of glia cells was in no great excess of the normal.

The vessels showed the following changes: The intima showed a distinct proliferation, though this did not result in the marked thickening so characteristic of congenital lues. The media was normal except for some cellular infiltration. The adventitia however was strikingly affected, especially in the way of infiltration. The adventitial spaces were very greatly dilated and were penetrated throughout by a close infiltration of round cells. These were of three kinds: (1) Small mononuclear cells; (2) larger mononuclear cells; (3) plasma-cells. These last were in considerable numbers.

In addition there was a cellular infiltration widely extending into the cortical substance. This mainly consisted of mononuclear cells, though a small number of plasma-cells were also present at a distance from the vessels. Every here and there, usually in the immediate neighbourhood of the vessels, the infiltration condensed so as practically to form a "miliary gumma."

One need hardly say that in the preparations no spirochætes were visible.

(c) *White matter.*—In general, similar changes were found here, though to a much less extent. The differences were of a quantitative and not qualitative kind. The nerve-fibres were tolerably well preserved.

Conclusion.—The above-mentioned changes warrant the inference that in this case we have in all probability to do with a subacute case of lues cerebri. A noteworthy feature was the excess of the cellular infiltration over the other changes present.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the* 13*th*, 1907.)

35. "INHERITED SYPHILIS."

By GEORGE E. SHUTTLEWORTH, M.D.

I FEAR my contribution belongs rather to the ancient history of the subject than to the era of bacteriological pathology in the light of which we now view the problems of inherited syphilis. Some twenty years ago I summarised (for a paper contributed to the Ninth International Medical Congress at Washington) the views held by such authorities as Mr. Jonathan Hutchinson, the late Dr. Langdon Down, of Earlswood, Dr. Fletcher Beach, then of Darenth, and my own experience in two large institutions for imbecile children, showing that in this country at any rate the *rôle* of parental syphilis was surprisingly small, so far as could be ascertained from a scrutiny of the histories and characteristics of the inmates of British imbecile asylums; that is to say, that it figured as an ætiological factor in less than 2 per cent. of the cases. In a later investigation of case-book records of 1200 patients who had been under my care at the Royal Albert Asylum (for an article on the "Ætiology of Idiocy," published* in 1892) I found the percentage with parental history or personal evidence of syphilis to be 1·33. Dr. Fletcher Beach, dealing with 1100 cases at Darenth on the same lines, found that his percentage worked out at only 1·01. Thus in an aggregate of 2380 cases at the two asylums, in which histories had been fairly well ascertained, we were unable to find evidence of the existence of syphilitic taint in more than 1·17 per cent. of our patients.

In a later investigation, made in 1898 by Dr. Telford Smith, my successor at the Royal Albert Asylum (with the aid of Dr. Coupland) for the special purpose of ascertaining the frequency with which inherited syphilis might be regarded as an ætiological factor in the 580 cases then resident in the institution, he found that only in three

* Hack Tuke's 'Dictionary of Psychological Medicine,' vol. ii.

cases was there an acknowledged history of congenital syphilis and only eight cases presenting stigmata of the disease.* He expresses the opinion, however, in which I fully concur, that although the percentage of syphilitic idiots and imbeciles found in asylums is extremely low, it does not follow that parental syphilis has but little to do with the production of mentally defective offspring, considering how high is the infantile mortality of children with this taint, and that as a rule institutions for imbeciles only receive those who have attained the age of five or six years.

More recently (viz. in 1902) Dr. Tredgold (L.C.C. Research Scholar in Insanity) made a careful examination of 150 juvenile "aments" in metropolitan asylums, in which he was able personally to investigate the family histories, and found amongst that number only four (=2·5 per cent.) in which syphilitic taint was manifested by characteristic signs, and five (= 3 per cent.) in which it was possible on the ground of ascertained parental infection, though the children themselves showed no specific physical stigmata.† Including the latter, the aggregate percentage would amount to 5·5, but Dr. Tredgold adds that in all these cases other ætiological factors existed, antecedent insanity in five of the cases, and in the remainder either phthisis, or alcohol, or both. It is probable that where other contributory factors exist, and notably a neuropathic heredity, the syphilitic taint exerts a pernicious effect on the mental integrity of the offspring, for, as Dr. Tredgold suggests, "the syphilitic poison has a special predilection for finding out the weak spot." It would seem, however, that its toxic action is not so effective in producing primary formative arrest of brain development, *i. e.* congenital defect, as in causing subsequent degenerative changes in brain and nerve tissues. Dr. G. A. Sutherland has, indeed, suggested that it is a frequent factor in the production of the "Mongolian" variety of congenital idiocy, but my own somewhat extensive experience of cases of this type does not lead me to concur in this view.

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* 'Brit. Med. Journ.,' October the 15th, 1898.

† 'Archives of Neurology' vol. ii.

Degenerative changes due to this cause may, indeed, manifest themselves early in life and give rise to cranial osteitis, meningeal inflammations and eclampsia, epileptic and paralytic symptoms so often associated with mental defect in children, and frequently assigned as its cause, though more correctly to be regarded as links in the chain of causation. But the most characteristic type of mental degeneration in the young associated with hereditary syphilis is that designated by Dr. Clouston in 1877 "juvenile general paralysis" (or by Dr. Judson Bury as "juvenile dementia"), in which a break-down (mental and physical) occurs at the period of second dentition or advent of puberty, leading to a fatal issue in a few years. Such cases are found not to be so rare in asylums as was formerly thought, and Dr. Mott has tabulated the histories of twenty-two in vol. i of the 'Archives of Neurology.'

It would appear from this table, as well as from a paper in the 'Practitioner,' January, 1908, in which an aggregate of forty cases are dealt with by the same author, that in at least 80 per cent. of these cases there was evidence either of syphilitic family history or of the presence of syphilitic stigmata (in several instances of both), and he states his opinion that "though there may be many exciting causes the predisposing cause of this disease (juvenile general paralysis) is nearly always hereditary syphilis," thus agreeing with the conclusions of Thiry, Alzheimer, Mendel and other continental pathologists, that inherited syphilis plays a predominant *rôle* in its ætiology. Dr. G. A. Watson, in an investigation of twelve cases published in 'Archives of Neurology,' vol. ii (p. 624), states that "in no single case could the effect of syphilis be excluded." This latter statement is borne out in the records of various cases that have from time to time been brought under the notice of this Society. Thus in vol. iv of the 'Reports,' p. 31, we find the case of a girl, aged 11 years, exhibited by Dr. George Carpenter, in which there were typical Hutchinson's teeth and a patch of left choroiditis, with a suggestive family history and the personal characteristics of general paralysis of the insane. In vol. v, p. 52, a case of juvenile general paralysis (a boy aged 11½ years) is recorded by Dr. James Taylor, who remarks that though the patient shows none of the undoubted stigmata of congenital syphilis, and has a family history free from suggestiveness, "yet the meagre physical development, the suggestive facies, and the Argyll Robertson pupils, if not conclusive, are sufficient to raise suspicions."

To discover the existence of syphilitic taint a careful scrutiny, not only of parental history, but also of brothers and sisters, may be necessary; for, as Dr. Mott observes in his recent article, "it is remarkable how often one found absolutely no signs of syphilis on the body of a juvenile paralytic patient suffering from general paralysis, whereas brothers and sisters showed well-marked signs."

Taking all the evidence into consideration, I am inclined to think that inherited syphilis is a more frequent factor in the production of mental defect and abnormality in childhood than can be demonstrated from the institution statistics I have referred to, and to agree with Fournier that many cases of impaired mental development, such as are met with in children relegated to special schools, have their origin in an inherited syphilitic taint, normal brain development having been interfered with by osteitis causing cranial thickening, by meningeal indurations or by localised cerebral sclerosis. But there is great need for more exact observations in these directions, and I trust that these and other pathological questions may be elucidated by those undertaking the medical inspection of schools.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the* 13*th*, 1907.)

36. "INHERITED SYPHILIS."

By G. A. SUTHERLAND, M.D.

I THINK we are sometimes hindered in arriving at a correct diagnosis of a lesion which is syphilitic by the fact that all the more common signs of syphilis are not present in the patient. It is not usual for a syphilitic baby to have about its person all the stigmata of congenital syphilis. If one system or one organ is very markedly affected the chances are that other systems or organs may escape, or be but slightly affected. A combination of syphilitic lesions which are visible certainly aids in the diagnosis, but in many cases the diagnosis, if made early, must be based on the appearance of one lesion. The age of occurrence is often a valuable help. A positive history of parental syphilis is useful, but a negative one is of no value. Treatment may prove a valuable aid in the diagnosis. By mercurial treatment some understand the administration of a quarter or a third of a grain of grey powder twice a day. This is an excellent stomachic remedy in some cases of indigestion, but its effect on a syphilitic lesion may not be manifested for some months. As a diagnostic agent mercury should be given in full doses—one to two grains three times a day. In the case of an active lesion a definite result will be recognised within one or two weeks. If the result is negative the drug need not be continued.

Syphilitic lesions of the bones during the first three months of life are common. They are: (1) syphilitic epiphysitis, (2) changes in the skull, and (3) osteogenesis imperfecta syphilitica.

The occurrence of pseudo-paralysis in a limb, coming on gradually, without marked fever or constitutional disturbance, and without any source of septic infection, is usually due to syphilitic epiphysitis. It yields readily to mercurial treatment.

The skull may show the characteristic changes of cranio-tabes, *i.e.* small areas of softening and absorption of bone in the occipital, parietal, and frontal regions. These areas are often so small as not to be palpable during life, but may be well seen post mortem, when the coverings of the skull have been removed. The changes around the fontanelle take the form of thinning of the bone, so that the margin may have an unusually sharp feel. This is in marked contrast to the thickening of the margins of the fontanelle which appear later as the result of rickets. General enlargement and thinning of the bones of the skull may follow as the result of hydrocephalus. Hydrocephalus, which is present at birth or develops during the first three months of life, is usually due to syphilitic meningitis about the base of the brain. Excellent results are obtained in many cases by the use of full doses of mercury.

One form of osteogenesis imperfecta would appear to be due to syphilis. At birth many of the long bones of the extremities may be found to be fractured, due to the mechanical strain during parturition, or possibly intra-uterine fœtal movements. While the fractures are usually in the shafts of the bone, there may also be epiphysial and subperiosteal effusion with marked swelling and tenderness of the limbs. Fresh fractures may occur after birth from merely handling the limbs. Fractures also are common in the ribs. The skull may show deficiency of bone formations in parts, and effusion under the pericranium. The condition is a very serious one, both from the severe nature of the syphilitic lesion, and from the weakening effect of the pain on the infant. Under full mercurial treatment a rapid improvement in the condition may sometimes be observed. The subperiosteal effusion subsides, the fractures unite firmly, and the only permanent trace of the illness is a distorted condition of the limbs from the badly united fractures.

(*Read at the special meeting for the discussion of "Inherited Syphilis," December the* 13*th*, 1907.)

37. ON CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS AND ITS MEDICAL TREATMENT.

By GEORGE CARPENTER, M.D.

For some time I have been on the watch for a clinical example of pyloric obstruction in an infant which would convince me as to its undoubted hypertrophic nature, and which would respond to medical treatment. The case I shall presently relate satisfies those requirements.

I have been well acquainted with dyspeptic troubles in infancy without pyloric hypertrophy which clinically give all the symptoms of that complaint except one important finding, and that is the pyloric tumour.

Cases such as these I, too, have had my successes with medically. But if I were to enumerate all my experiences in that respect, then I also would be able to bring to your notice a magnificent array of cases of congenital hypertrophic stenosis of the pylorus in place of this solitary example of a successful case medically treated.

It is but the other day, figuratively speaking, that serious attention was drawn to this condition, and the great number of cases that have fallen to the share of, and have been recorded by, some observers, must be due to the inclusion in their statistics of the functional with the organic variety, of the very common with the rare, for it cannot be allowed that hypertrophic stenosis is any other than a rare condition.

On the other hand, infantile cases of functional gastric disturbance with symptoms suggesting pyloric stenosis are, comparatively speaking, as common as blackberries on the brambles in autumn.

Some such explanation must be held accountable for the high percentage of cases which have fallen to the share of some writers on this subject in so short a time.

But I must confess that I did not think it possible to cure such examples of congenital hypertrophic stenosis of the pylorus as have come within my clinical experience without surgical aid.

In the cases of that complaint that I have treated the pyloric tumour was hard like gristle, cut like the cartilage of the knee, was felt through the abdominal wall, the infants were operated upon, they all died, and their stomachs were exhibited to the Society in due course.

The case that I am about to relate presented precisely similar clinical symptoms and identical physical signs, was by reason of its wasted and poor condition of vitality unfit for surgical operation, and would like them, I have no doubt, have died if it had been operated upon.

It is remarkable how many recorded cases there are where no pyloric tumour was felt and where the typical appearance was found either at operation or in the post-mortem room, a sure sign, however, that the diagnosis was correct.

But notwithstanding these repeated failures to detect the tumour during life, I must confess that the diagnosis of congenital hypertrophic stenosis of the pylorus does not satisfy me unless the tumour be felt.

Perhaps the failure to find it may in some cases have been due to want of experience of what to feel for. But I think if greater care had been exercised in making the abdominal examinations the little tumours would not have so frequently remained undiscovered. But granted that due care had been taken, I think I can offer another explanation for failure to feel the tumour.

By the usual method of examination—that is, by palpation—the tumour is apt to recede from the fingers and to drop deep down into the abdominal cavity. Now, if in addition to the usual method of abdominal exploration the examiner grasps the abdominal parietes and the underlying parts with the fingers and thumb from right to left and *vice-versâ*, he will the more readily find the characteristic small hard mass, and not only detect it, but gauge its size and shape and even note the accompanying induration of the stomach walls. In thin babies no difficulty should be experienced, and a plump healthy infant with this condition is not likely to come under medical observation.

Prior to my own experience about to be related, although preserving a perfectly open mind on the subject, I doubted whether the true explanation had been arrived at for the pyloric tumours which had been found in the cases treated successfully medically. Indeed, on perusing some of the recorded examples of congenital hypertrophic stenosis of the pylorus in which a tumour is stated to have been felt, I am still of the opinion that they were not correctly diagnosed—that is, if the account given of the cases accurately describes what was found clinically. An enlarged gland had already once been mistaken for a thickened pylorus, why not again? Or why not some other pathological condition? But now I no longer have any doubt, and I am perfectly satisfied that cases of congenital hypertrophic stenosis of the pylorus can be relieved by medical means and without the surgeon's aid.

Hopeless in regard to medical treatment as the typical hypertrophied pylorus appears to be in the post-mortem room—the tightly shut orifice, the thick gristle-like walls, the cervix and os uteri-like appearance on its duodenal aspect, its capacity to securely hold a dilated stomachful of water—I do not think this after-death state denotes its natural condition during life.

I think tight post-mortem contraction of the hypertrophied circular muscular fibres occludes the pylorus, and this prevents the exit of water when tested in the usual way. Indeed, quite apart from other reasons I know that this must be so, because I have seen the channel patent (No. 4 catheter), during life, even in an extreme case of this interesting condition.

There are two hypotheses as to the causation of congenital hypertrophic stenosis of the pylorus, viz.: (1) That this condition is a muscle hypertrophy not only of the pylorus but of the rest of the stomach, and that it is secondary to a functional disturbance (Heubner*); (2) That it is a primary malformation or new growth. Heubner does not state definitely whether his conclusions were based upon his own post-mortem experience; in any case he could not have performed more than one autopsy as he had only one death. And secondly, from the remarks in his paper, which was read before the Children's Section of the Seventy-eighth Versammlung Deutscher Naturforschen und Aertze in Stuttgart, he gives the impression that he bases his conclusions on autopsies performed by Wernstedt. He claims forty-nine cases out of a clinic of 10,000 children.

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* 'Monatschr. f. Kinderheilk.,' Bd. 5. S. 355, 1907.

In regard to the two views I am in favour of the congenital hypothesis for these reasons:

(1) The condition has been found in the fœtus (Dent); (2) symptoms frequently arise immediately after birth in cases where it is known to exist; (3) the associated muscle hypertrophy of the stomach is not a general hypertrophy of the organ, or if it be a general hypertrophy of the viscus the part near the pylorus, to the extent of a quarter or one third of the pyloric end, is immensely thicker in comparison.

The stomach being a hollow viscus I think hypertrophy would be general rather than partial if that hypertrophy were due to increased work. It is so, as a general rule, in the bladder with a large prostate, the nearest resemblance to it that I can think of, whatever may be the exceptions to that rule, if there be any.

I have made microscopical examinations in three cases, and I have found in the pylorus thickening of *all* the muscular layers, including the muscularis mucosæ.

The longitudinal fibres were two or three times larger than they should have been, but they were not hypertrophied to the same comparative degree as the circular, which were very powerful. Further, there was an increase in the fibrous tissue, and the submucous layer was larger and thicker than normal. The mucous membrane was healthy.

The thickened pylorus ended abruptly at the duodenal side (cervix uteri-like appearance), but gradually tapered off into the stomach walls on the opposite side. On the outside it was constricted in the centre.

In the stomach of one case all the layers were hypertrophied, but especially in the pyloric fourth, and the circular fibres were equal to the two others combined. In the others there was considerable hypertrophy of the stomach walls of the pyloric third or fourth. The submucosa was thicker than natural. The mucous membrane was natural.*

There is not a great amount of information to be obtained from the literature on the microscopical side. One writer † states that in his case the submucosa showed fibrous thickening in addition to hyperplasia of the circular muscular coat. In a remarkable case, published by Audry and Savonnet,‡ the pylorus was 1⅛th inch long and nearly ¼th inch thick, very hard and incompressible, and of normal colour. The stomach was not dilated but slightly hypertrophied.

The pyloric stricture was found to be caused by hypertrophy of the muscular coat. Histologically signs of subacute inflammation were evident. In the interstices of the gastric muscle, of the pyloric ring, and especially of the most exterior longitudinal fibres, there were groups of fusiform and round cells. The submucosa and the muscularis mucosæ were also much infiltrated with fusiform cells. Each blood-vessel was ensheathed with a double row of cells. The glands and the interglandular spaces were too rich in round cells. There was hypertrophy, but of a very evident inflammatory kind.

In regard to naked-eye appearances the stomach may or may not be dilated, and it is not necessarily dilated in even pronounced conditions of the affection. On the other hand it may be very large. Peristalsis may or may not be seen. This occurs in the undilated as well as in the dilated organs, and is best seen when the infant is marasmic, and it is a late, not an early, symptom.

Although symptoms in cases of hypertrophic stenosis often start from the birth, yet this is by no means invariably the case, and sometimes it is one, two, three, four or more weeks before their onset.

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* 'Reports of The Society for the Study of Disease in Children,' vol. v, p. 242, 1905; vol. vi, pp. 27 and 30, 1906.

† J. Dorning, 'Arch. of Pediat.,' September, 1904, p. 681.

‡ 'Lyon Méd.,' May the 7th, 1905.

The infant then commences to vomit. If this be not checked other symptoms rapidly supervene, and steady deterioration results. When vomiting occurs in grim earnest, constipation and scanty urine are its associates. Vomiting may be both irregular as to time and in quantity; the vomit may be small or consist of accumulated feeds. There may be nothing special about the act or the stomach contents may be pumped up violently on to the floor. In a well-marked case bile is absent from the vomit. The stools may be meconium-like, sage green, dark green, dark brown, or tarry. But even these may disappear and be followed temporarily by natural motions. In some cases the symptoms that attract most attention are constipation and marasmus with a dyspeptic background.

Prior to all these symptoms, however, it often happens that the child has enjoyed good health, and there has been no occasion to suspect any organic gastric malady. Perhaps it has been taken from the breast and put on the bottle, and then its troubles began.

Although the infant in such cases has displayed no symptoms, I do not think that there is any reason to doubt that the thickened pylorus has been comfortably tucked away all the time.

Granted that this be so, why does it suddenly become troublesome?

Cases with classical symptoms of stenosis of the pylorus—the expulsive vomiting of accumulated feeds, meconium-like motions, scanty urine, and peristalsis—have succumbed to the malady, and post-mortem the pylorus has been found normal.

The explanation that has been given for cases such as these is that they were suffering from pyloric spasm.

Granted that pyloric spasm can kill an infant with a healthy pylorus, it can as readily accomplish the act with a hypertrophied pylorus.

Graft the spasmodic affection on the organic and the explanation of the symptoms is at hand.

Bloch, of Copenhagen,* is under the impression that active symptoms in his cases were produced by gastric catarrh, because he found mucus and pus corpuscles in their stomach washings. This, however, will not explain all the cases—mine, for instance—because I found the mucous membranes of their stomachs normal.

Perhaps the over-powerful strongly-acting pylorus causes congestion or œdema of the overlying mucous membrane with blocking of its lumen, or there may be other explanations for its local congestion. It may have its analogy with the Saturday night reveller who, after his potations, suddenly finds himself with retention of urine due to congestion of the mucous membrane covering an urethral stricture.

Judged by the stomach irritability displayed in these cases, the speedy response by waves of gastric peristalsis of regular or irregular rhythm to even so little as the lightest tap over it it is likely that the pylorus also is in an equally excitable state.

It may be that under such circumstances the mere mechanical irritation of the food tends to keep the pylorus in a constant state of reflex spasm, let alone any toxic action on the muscles or nerve centres that might be supposed to arise from the complex chemical products retained in the stomach.

But whatever may be the explanation of the obstruction at the pylorus, I have no doubt the causes for it are complex and multifarious—complex and multifarious as are the causes for gastric disturbances that occur in the dyspeptic infant, and it is the dyspepsia that is at the bottom of it, and that is the important factor in these cases.

This is not based solely on hypothetical conditions, for have we not pathological evidence to support it. A suckling under Bloch† was completely cured after having had four weeks' symptoms of congenital pyloric obstruction; he succumbed six months later to an attack of acute enteritis. The pylorus was found just as hypertrophied and just as constricted as in children who die of pyloric stenosis.

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* Quoted by R. Romme, 'Jahrb. f. Kinderheilk.,' 1907, vol. xv, p. 477.

† *Loc. cit.*

A study of the vomited matters has thrown no special light on the condition. In some cases there has been no free hydrochloric acid, in others this has been present, in others the vomit has been "intensely acid and sour smelling," and again in others mucus and lactic acid have been detected, but no free HCl.

Dismissing the question of hypertrophic stenosis of the pylorus from the mind for the moment and turning to the infant with a normal pylorus, we are daily faced by the problem of how to cater for infants with week digestions.

It is occasionally brought to our notice that the ideal infant food, the mother's milk, is not always the innocuous baby-growing material that it is supposed to be—quite the opposite.

There are some infantile digestions that cannot tolerate mother's milk, be it that of the child's own mother or that supplied by foster-mothers—not one foster-mother but a succession of them. They become sick, waste, and would die, and yet these same infants straight away make progress on a preparation of cow's milk that one would not have advocated on theoretical grounds. These are cases where sometimes the doctor does not score—the outsider, vulgarly speaking, "wipes the doctor's eye." Call it idiosyncrasy, what you please, the fact remains. There can be no question that some milks are toxic in their action, be they breast milk or cow's milk, and feeding with them brings about reflex muscular action and all the symptoms of congenital hypertrophic stenosis of the pylorus.

Dyspepsia in infants is a thing to be avoided, and infantile dyspepsias are often manufactured by the ignorance and stupidity of those in charge of them.

Once thoroughly start an infant on a downhill course of indigestion and the dyspepsia that ensues is prolonged.

The feeding of some infants is a constant source of worry and anxiety, and taxes all the resources and gastronomic ingenuity of the practitioner, and that under quite ordinary conditions, and even then success does not invariably crown his efforts.

If the feeding of the normal infant is a problem in some cases, it becomes additionally so if there happens to be hypertrophic stenosis in the background. If the normal dyspeptic infant does not always readily respond to medical treatment, and we know that it does not do so, it cannot be expected that the abnormal infant will react as, or more quickly than, a child with a properly formed pylorus.

In the cases we are treating daily without this deformity, we are prepared to struggle on and do our best to relieve symptoms. There is no reason therefore why despondency should set in when treating cases of hypertrophic stenosis of the pylorus because they do not quickly answer to treatment. They must be looked upon as difficult medical cases from the outset and dealt with accordingly.

Striking as have been the triumphs of surgery in the past in the treatment of this complaint, yet the mortality rate has proved a very heavy one.

The records show that the infant does not readily tolerate plastic operations on its abdominal viscera, even at the hands of the most skilful and the most expeditious surgeon. Nor has the medical treatment hitherto been anything to make a boast about. Nevertheless we must for the future, I think, look upon surgery as a *dernier resort* and not as the sheet anchor of treatment. It is hard upon the surgeons, I admit it, to ask them to galvanise into life a moribund athrepsic infant, and failing in that to take a share in the responsibilities and in the obsequies, but I think in the interest of the patient the surgeon must be thought of late in the case. But once let the physician realise that these cases are not untreatable medically, and there will in the future be more medical successes and fewer surgical failures. What the future will have in store for the cases of hypertrophic stenosis that have been cured medically remains to be seen. But in regard to this point it is not uninteresting to learn that Seefisch found that in three cases that responded to this treatment operation became necessary before the children reached the age of twelve years.

Surgery as a mode of treatment for pyloric spasm is neither necessary nor advisable. To operate on such a case would be on a par with performing tracheotomy for the cure of laryngismus stridulus.

I therefore think the following advice, which I read some little time back, in regard to the treatment of hypertrophic stenosis is calculated to do much harm. It runs: "If the treatment is to be successful the diagnosis of the condition must usually be made in the absence of two of the classical symptoms, viz. peristaltic movements of the stomach and the presence of a tumour." Nor can I on like grounds agree with the views of another observer who wrote: "Probably I do not hunt for the pylorus with the same care as others, as it does not appear necessary to do so, and the proceeding is very uncomfortable for the infant."

It is for the purpose of preventing unnecessary operations as well as from the desire to correctly diagnose the case that I attach very great importance to the discovery of the pyloric tumour, and the discomfort that a baby may suffer from such an examination will be a trifle light as air in comparison with a major surgical operation.

Had more attention in the past been paid to the detection of the tumour there would have been fewer cases on record of congenital hypertrophic stenosis.

Finally, what should be the treatment? There is no royal road to success in the treatment of dyspepsia without a pyloric tumour, and a similar favoured route must not be hoped for in the dyspepsia that is complicated by this condition.

The treatment must be conducted on the broad, general principles that experience has taught us to be of value in the uncomplicated complaint. Muscular and nervous hyperexcitability of the organ may be fought by antispasmodics, such as cocaine, opium, chloral, bromide, antipyrin, belladonna, camphor and sulpho-carbolates. I have no special partiality for any of these drugs, and I have no supreme faith in their performances.

The secret of success in all medical treatment of dyspepsia in infants lies not so much in its drug treatment as in the discovery of the milk best suited to the child. When bottle-feeding is compulsory the selection of just the right form and the right dilution of cow's milk requires careful study, but above all things it must be *clean.* A baby's stomach will deal efficiently enough with a poor milk, but it will immediately resent the stale and the dirty article. Therefore when feeding these cases the selection of a clean milk is no mean part of the programme. And not only should it be clean, but it should be undoctored. The food that is selected should be one that leaves a small residue—one that the gastric juice can readily dissolve. Large lumps of curd in the stomach are fatal to progress.

Regular meals are no less important than restricted meals, and not only are meals that are too bulky to be carefully avoided, but the meal must be administered slowly. The dyspeptic infant becomes an inveterate food-bolter, and a meal that is dumped down into the infant's stomach is bound to disagree. The man who is in a hurry to change the dietary because the infant's stomach does not immediately respond to his treatment will never succeed. Success can only be compelled by shaping a course and intelligently sticking to it. It will be found on enquiry that those who claim that they have tried everything have really tried nothing. Ringing the changes on a variety of foods invites dyspepsia, it doesn't cure it, and such panic-bred dietaries must be avoided.

Abdominal massage in the direction of the pyloric outlet has proved efficacious in at least one case. But in regard to topical treatment gastric lavage has a well-merited reputation. Thorough stomach washing with warm normal saline solution should be used twice a day until the complaint shows decided signs of improvement, when it may be tried once a day. That the remedies are working satisfactorily can be seen by the improved condition of the stools. And there are two other precautions that must be taken, viz. to keep the infant's stomach warm and not to upset its digestion by starchy foods.

But when is the surgeon to be called in? In the present state of knowledge, to assert that the surgeon should never be called in is perhaps not warranted by the fact, but nevertheless I think the advice should be hardly ever, and that the physician will best serve his patient by not being in too great a hurry to shift his responsibility on to his surgical colleague.

On May the 22nd, 1907, I saw R—, an infant, aged 2 months, in consultation with Dr. D. J. Munro, of Brixton. The history given was typical of the condition of pyloric stenosis—it kept down two or three feeds and then vomited what appeared to be the whole amount previously taken. It was constipated. It had gradually lost ground from the first week of its birth; in spite of being fed on a variety of foods it "vomited everything." Various proprietary foods were tried, milk and water, peptonised milk, but all with the same result: persistent vomiting followed, accompanied by constipation and passage of scanty concentrated urine. At a week old it was breast-fed, took well, weighed about 7 lbs., and did not vomit, but owing to the mother suffering from a slight attack of rheumatic fever the child had to be artificially reared, with the result just related.

The infant was very emaciated when I saw it, and seemed so ill that it did not appear that it would survive the night. Its pulse was slow and feeble, the respirations very shallow, the anterior fontanelle depressed, and the temperature subnormal. It was with great difficulty that it was kept warm, although it was swathed in cotton-wool and packed round with hot-water bottles. It was too weak to cry—it opened its mouth as though to cry, but no sound came from its lips. It weighed 5 lbs. 13 oz. The vomiting was now very persistent and violently expulsive, and the bowels almost absolutely confined.

An abdominal examination left no doubt in my mind as to the nature of the complaint. There was an indurated pylorus of the most typical feel. The lump was located above and to the left (examiner's) of the umbilicus, it was deep down in the abdominal cavity, it could be grasped between the fingers and thumb on deep manipulation, was as large as an almond-nut, and it felt as hard as gristle. Further, there was, on abdominal manipulation, obvious gastric peristalsis terminating in the direction of the lump aforesaid. Otherwise there were no physical signs; but there was a right inguinal hernia which had appeared a week after birth.

The physical condition of the child was so weakened that surgical measures could not be undertaken with a light heart. It was, however, pointed out to the parents that surgical operations had been performed on these cases and with perfect success. On the other hand, that cases had come under my notice where the condition of the pylorus was identical with that in the present instance and in which speedy post-surgical death had taken place. But there were also cases of recovery on record in which *medical* measures had been adopted. In the present case it appeared to us that submitting the child to surgical operation was tantamount to signing its death warrant.

It was therefore decided that the infant should be medically treated, that the diet should be whey, and that the stomach should be washed out daily. On the night of my visit Dr. Munro washed out the stomach with a 0·5 per cent. solution of common salt, this was followed by a 4 oz. feed of whey, and subsequently 2 oz. feeds every two hours were administered.

The day following and on subsequent occasions the wash was made of Na_2SO_4, 0·5 per cent., and $NaHCO_3$, 0·2 per cent., which had the immediate effect of causing regular bowel actions. Four-hourly drop doses of laudanum were also ordered. There then had been no sickness for twenty-four hours, and 14 oz. of whey had been retained. Inunctions of cod-liver oil were also instituted twice daily.

On May the 24th, the second day of treatment, the infant was sick twice following cod-liver oil by the mouth, but it retained 15 oz. of whey. He also passed two dark green offensive motions, the first for four days. The next day he was ordered tea-spoonful doses of olive oil by the rectum, and in place of the drop doses of tincture of opium (which were making him much too somnolent) half-drop doses were substituted combined with drop doses of tincture of belladonna.

The injections of olive oil were mostly all absorbed, but sometimes they were returned with a loose green stool and often undigested. The bowels were opened about once daily, and the motions were mostly small, green, and constipated.

From May the 23rd to June the 2nd the infant retained daily the following quantities of whey, viz. $14\frac{3}{4}$, $20\frac{3}{4}$, 19, 15, 18, 19, 17, 16, $16\frac{1}{4}$, and $17\frac{3}{4}$ oz.

On the fourth day after commencing treatment it had lost 2 oz. in weight, but its subsequent gain in weight amounted to 7 oz.—6 oz. during the last five days.

It was sick twice on May the 25th, three times on the 26th, twice on the 27th, twice on the 28th, and once on the 29th, and once on the 30th, and then not again for eight days, when on June the 7th it vomited twice.

It was fattening visibly. Such was its condition on June the 7th, but for several days it had suffered from erythema and œdema, and the possibility of this cutaneous disturbance being due to the olive oil injections occurring to Dr. Munro they were discontinued. But the rash still continued to appear for three or four days, so the rectal injections were re-commenced but then given once daily only. According to the nurse's description the rash "seemed to appear just after the olive oil injections." The lips became swollen, red patches appeared over the face, rather like port-wine stains; the hands and feet were red and swollen. Besides the swelling of the hands and feet swelling was noticed on the right side of the sacrum; it was, quoting the nurse's words, "quite dropsical," and about two days later spread over the body. The infant increased in weight—an increase which was thought to be only owing to the swelling just mentioned. By June the 5th there was a good deal of dropsy of the neck; he coughed a lot over his feeds; there seemed a great difficulty of swallowing and also breathing; he was propped up with pillows, and often had to be held up by his feet as he choked and "went black in the face" while being fed. The œdema appeared to be accountable for these troubles.

On June the 6th the opium was discontinued (tr. digitalis $1\frac{1}{2}$ ♏ being substituted); the infant then became brighter and cried more after its discontinuance.

On June the 7th I again saw the infant with Dr. Munro; œdema was at that time a marked feature, though less extensive than it had been. The erythema had disappeared. The urine contained 2 parts per 1000 of albumin, but there were no casts; it was slightly alkaline when passed, and deposited a slight precipitate of earthy phosphates. Dr. Munro subsequently examined the urine, and came to the conclusion that there was no deficiency of chlorides. Dr. Munro reported on June the 11th that the œdema had quite disappeared since omitting the laudanum. Magnesium sulphate, ·5 per cent., was now used by him for lavage, with the idea that the magnesium base would assist in the elimination of chlorides.

Starting on June the 8th he was fed on "humanoid" milk, an Aylesbury dairy company product. He took it well, but only in small quantities; fifteen grains of cane sugar were added to each feed. He appeared to be more satisfied with the new diet, and he slept well, but the weight fell to 5 lbs. $12\frac{1}{2}$ oz. It was on this date intended to wash out the stomach every thirty-six hours in place of every twenty-four hours as was previously the case.

On June the 14th Dr. Munro reported that he was taking and digesting well, but "only in small quantities, rarely more than 2 oz. per feed." He was still losing weight, having dropped to 5 lbs. 7 oz.—less than ever. He was sick while being fed once, the first time for a week, there having been no sickness since the 7th.

He was ordered a milk-and-water mixture—one part milk and two parts water every two and a half hours—2 to 3 oz. at a feed, and to three of the bottles a teaspoonful of cream was added. On the 15th, as the mother was anxious for it, a wet nurse was obtained and mixed feeding was instituted. The infant was sick once on the 16th and three times on the 18th; on this latter occasion coffee-ground vomit occurred, which was thought to be due to cracked nipples. The wet nurse not being a success was discontinued. Lavage was discontinued on the 20th. It was sick once on the 21st, and not again until the 28th, when it was sick once and then for the last time.

During the first week of milk and water diet it lost 5½ oz.; after that it commenced to forge ahead and gained on an average 1 oz. a day. It suffered a good deal with flatulence all through its illness, especially after food, and sometimes would cry nearly all day, as if in pain. (After wearing a woollen truss, which was applied in the middle of August, it cried ever so much less.) It perspired a good deal at night.

On July the 6th it went to Silchester.

Its weight on June the 14th, when milk-and-water feeding was started, was 5 lbs. 7 oz., and on July the 8th it was 6 lbs. 12 oz. Day by day it took increasing quantities of milk and water; on June the 14th the amount was 18 oz., while on July the 6th it was 30 oz. While on whey the bowels were opened once daily, and the motions were green, but after it had been on milk and water for a week the stools became quite natural, and there were from two to four motions in the twenty-four hours.

Week by week it continued to gain ground, and by July the 27th, when the rectal injections of olive oil were discontinued, it weighed 8 lbs. 1 oz.

On August the 22nd, when it returned to London, it weighed 9 lbs. 8 oz., and on August the 27th, when the nurse left it, it weighed 10 lbs. and was steadily gaining ground every day. In the middle of September it suffered from spongy tender gums for a week, but since impressing the necessity for antiscorbutic foods upon the parents it has never looked back. In October the infant weighed over 16 lbs. at seven and a half months, and was a jolly, normal baby, and he still (January, 1908) continues to thrive.

(*Read January the* 17*th*, 1908.)

38. THE PATHOLOGY OF CONGENITAL HYPERTROPHY OF THE PYLORUS IN RELATION TO TREATMENT.

By EDMUND CAUTLEY, M.D.

Such widely divergent opinions have been expressed on the treatment and pathology of the affection known as congenital hypertrophic stenosis of the pylorus, infantile pyloric hypertrophy, or congenital gastric spasm, that I have thought it worth while to put before you a few observations and comments on the pathology of infantile pyloric obstruction and the manner in which a cure may be brought about by medical treatment. Without a plausible explanation of the relationship of the treatment to the pathology it does not appear possible to estimate, with any approach to accuracy, the value of medical treatment or the necessity for surgical interference. On the one hand it has been stated that no case will recover under medical treatment; and on the other, that surgical measures are unnecessary, and that all cases will get well if treated by careful lavage and dieting. No doubt the truth lies between these extremes, and it is probable that the extremes really represent two different affections.

My own views are well known to you. I believe that there are two different diseases :

(1) Pyloric spasm.

(2) True pyloric hypertrophy, which may or may not

be associated with spasm or secondary effects, and may be moderate or excessive in extent.

In fatal cases and in those submitted to operation the naked-eye evidence of hypertrophy is conclusive proof of the accuracy of the diagnosis. When recovery ensues no such absolute proof is available. A trustful reliance on dilatation of the stomach, peristalsis, and even a palpable pyloric tumour is not always warranted. I have known cases in which the pylorus was said to be distinctly palpable and yet no hypertrophy was found after death.

It is interesting to observe that ten years ago the existence of this hypertrophy was not readily admitted, and that, by those who recognised the affection, it was regarded as extremely rare and difficult to diagnose. Now, the statements of some physicians would lead one to suppose that the affection is very easy to diagnose and remarkably common. My own experience confirms the first view. I have seen eighteen cases in ten years, and in spite of the anxiety with which I look out for such cases, only one of them has been under my care in the last year, and a further case seen in consultation after operation, which is not included in these figures.

The pathology must be considered in reference to spasm, hyperacidity, hyperplasia, and secondary effects such as gastric catarrh, œdema of the pyloric mucosa, and gastric dilatation.

Pyloric spasm.—Is there such a condition as pyloric spasm? Can it give rise to hypertrophy or can it exist for a long time without causing hypertrophy? Are the symptoms due to pyloric spasm liable to be mistaken for those due to hypertrophy? There seems little doubt that pyloric spasm can occur at all ages, and that it may prove fatal in infants without causing hypertrophy. Such a case I have already reported to this society (vol. vi, p. 39). The babe, aged 11 weeks, was admitted into hospital under my care for supposed hypertrophy of the pylorus. The vomiting had existed since birth, and was extreme. It ceased while in hospital, but death ensued on the day after admission, and no hypertrophy whatever could be found after death. I have seen many similar cases which have ended in recovery under medical treatment and in which there was no pylorus palpable during life.

On theoretical grounds I am inclined to maintain that pyloric spasm does not cause hypertrophy. There is no analogous hypertrophy in other parts of the alimentary canal as the result of spasm. Prolonged anal spasm does not cause it. Provisionally, until our knowledge of the subject is more certain, I accept the view that the action of the pylorus is similar to that of a closed door, which opens in response to certain stimuli. The pylorus is normally in a state of contraction, and only dilates to allow the passage of the contents of the stomach in response to stimuli received from the duodenum, dependent on the reaction of the duodenal contents. If this is the physiological action of the pylorus, it is barely credible that the amount of spasm sufficient to oppose the stimulus to dilatation can be great enough to produce the excessive hypertrophy of the circular muscular fibres so constantly present. There are both circular and longitudinal muscular fibres in the pyloric sphincter, but the hypertrophy is practically limited to the circular fibres, or, at any rate, the longitudinal fibres are relatively very little increased in bulk. If the longitudinal fibres possess the power of dilating the pyloric sphincter, how is it that they do not show marked hypertrophy as the result of pyloric spasm? If they act as opponents of the circular muscular fibres, one would expect that they would undergo a degree of hypertrophy proportionate to the hypertrophy of the circular muscular fibres. There is no such hypertrophy. It must be admitted that pyloric spasm may occur at any age, and that it is not an affection peculiar to the first few months of life. If this be the case there should be proof available of hypertrophy associated with spasm at all periods of life, that is, in cases in which there is no history pointing to pyloric trouble dating from early infancy, and we must admit also that it can occur *in utero*, for hypertrophy has been found in a seven months fœtus.

The cause of spasm is uncertain. It is generally ascribed to hyperacidity. Possibly it may depend on the toxines of decomposing food or result from dilatation. Using Toepfer's reagent (not a reliable indicator of hydrochloric acid) Knöpfelmacher found hyperacidity. It is probable that it can occur, though rarely, in infancy. Freund claims that he has cured pyloric hypertrophy by a diet of undiluted cow's milk. Such milk combines more readily with hydrochloric acid than does breast-milk, and hence there is less free acid. In the breast-fed child free acid appears in the stomach in about an hour and a half. On a diet of cow's milk it does not appear for two hours or more. R. Miller and W. H. Willcox ('Lancet,' 1907, vol. ii, p. 1671) have recently published some valuable observations on the gastric secretion in three types of cases in infants. In marasmus or atrophic dyspepsia the acidity and ferment activity of the secretion were diminished. There was no mucin and no retention of stomach contents, and the stomach was nearly emptied in thirty minutes after a test meal. In five cases of hypertrophic stenosis, four of which were verified after death, they found much mucin, marked retention of stomach contents, marked increase in ferment activity, and variable acidity with a tendency to diminution, probably dependent on the degree of gastritis, and, therefore, due to the secondary effects and absent in early stages. All these results were modified by systematic lavage, but the fatality does not appear to have been influenced thereby. In the third type, pyloric spasm or acid dyspepsia, they found no mucin, no retention of food, increased acidity, and normal or subnormal ferment activity. A dilated stomach might be seen to "stand up" on tapping, but there was no true peristalsis and no palpable tumour. They found further that if the gastric juice from a case of pyloric hypertrophy was added to milk, it caused firm and rapid clotting, and that milk was consequently a bad diet in such cases—a result diametrically opposed to Freund's observations. These patients with hyperacidity, especially when bottle fed, are liable to be diagnosed erroneously as suffering from pyloric hypertrophy. Dilatation of the stomach is common in the bottle-fed, and, if it is associated with wasting and pyloric spasm, it is obvious that a dilated viscus can be mapped out and that a contraction, suspiciously like the peristalsis of true hypertrophy, may be obtained on tapping. The vomiting may be similar in character as well.

The hyperplasia theory.—The other view of pyloric hypertrophy is that it is a congenital hyperplasia. It is possible that Nature, in her extreme anxiety to create an efficient pyloric sphincter, has over-exerted herself and produced too great an amount of muscular tissue. Perhaps it is a reversion to an ancestral type of stomach. The occurrence of this hypertrophy in a seven months fœtus has already been mentioned. If this solitary case be regarded as little evidence, I would point to the fact that all these cases occur within the first three or four months of life, that the symptoms generally arise within the first three weeks, and that often they begin as soon as the child is put to the breast. They occur in the breast-fed more frequently than in the bottle-fed, probably because the majority of infants are at first breast fed. The early onset of symptoms is to my mind absolutely a proof of an ante-natal origin, but I am not prepared to accept Dr. John Thomson's theory of intra-uterine spasm dependent on inco-ordination, or that such spasm or inco-ordination can produce hypertrophy. The theory of hyperplasia affords a plausible explanation of the course of the disease, the variability in the degree of hypertrophy, and the possibility of curing some cases by medical treatment. At birth there is a pylorus which contains an excess of circular muscle-fibres, but the canal is patent, or may be so, sufficiently to permit the exit of enough food from the stomach to maintain life and provide for growth. If it remains in this state, and the food is of a character to pass through with ease, there may be no symptoms. In some cases normal stools are passed during the first week or two of life. Usually the circular muscle gradually contracts and narrows the lumen, or it becomes so rigid that it is unable to dilate sufficiently for the ready exit of the stomach contents. Then the stomach slowly dilates and the characteristic symptoms of a dilated stomach develop. Sometimes the symptoms are very slight at first and then become so acute that everything is vomited, and the ejecta may be bloodstained. This may be due to secondary gastritis, or an acute œdema of the pyloric mucosa, the result of the contraction interfering with the local circulation of blood and lymph. It is very noticeable that in such instances the pylorus is found at operation intensely congested, almost purple in colour.

It seems to me that this theory best explains the various types of the disease which come under our notice. Thus we may find a palpable pyloric tumour, little or no vomiting, and no evident dilatation of the stomach. These patients may continue for days or weeks without definite or characteristic vomiting, even though not treated by lavage. They may even gain considerably in weight when suitably fed, and possibly get well, if the pyloric canal does not undergo further constriction and none of the secondary effects arise. My own experience is unsatisfactory. Three out of my last thirteen cases have been of this type. They were not submitted to operation, they gained weight at times, and they eventually died. In another variety there is definite dilatation of the stomach. Here, too, I am prepared to admit the possibility of recovery without surgical interference, provided that the dilatation of the stomach can be cured and the degree of obstruction is only moderate. Some of these have associated gastritis, on which the acute symptoms depend. This may improve on careful dieting and systematic lavage. In the cases of the above types fæcal matter is found in the stools, though not always if there is much gastritis.

In the more severe forms, some of which are extremely acute in their development, the stools contain no fæcal matter and consist of bile, mucus, and epithelial *débris*. Vomiting is marked, often characteristic, and peristalsis is evident. Dilatation develops in a short time, but it is not advisable to wait for its development in order to clinch the diagnosis. The pylorus cannot always be felt, for the baby may be quite fat. Even in marasmic infants it is not always possible to be certain that one can feel the pyloric tumour.

The medical treatment.—If medical measures are curative there must be a rational explanation of their mode of action. Drugs appear to be useless. Dieting consists in giving a food which is as little irritating as possible, and contains little solid material. Whey and albumin-water fulfil these indications. Breast-milk is suitable, but many cases have occurred in breast-fed infants. Such a diet can only prove beneficial because it is able to pass through the unduly narrow pylorus or by preventing spasm. If the spasm is due to hyperchlorhydria undiluted cow's milk or a food rich in proteid ought to have the best effect. Some of the milk-proteid preparations might be tried. I have not found undiluted cow's milk retained in cases of true hypertrophy. Whey or albumin-water would be suitable if there is simply narrowing of the pylorus without secondary effects. It may prove beneficial in the reduction of gastritis. It may prevent spasm if the spasm is due to indigestible curds of milk and not to excess of acid. To cure the child it must be sufficiently nutritious to maintain life and provide for growth. At first it often produces remarkable gain in weight, for the child has been previously suffering from starvation. Usually the good effects only last for a week or two at most, and then the symptoms reappear, or the child ceases to gain weight, and any increase in the strength of the food at once induces vomiting. Hence it appears that diet alone may be curable in cases of pyloric spasm, and possibly in a few mild cases of true hypertrophy.

The other great remedial measure advocated is lavage. It seems to me that the exponents of this treatment must advocate it on the grounds that it clears the stomach of irritating contents, and so prevents spasm—that it reduces the gastric dilatation and cures the gastritis. Undoubtedly the vomiting varies directly with the frequency of the lavage, for the stomach is kept more or less empty and freed from irritating contents. If the case is one of simple spasm, due to stomach irritation, lavage is obviously a simple measure and likely to prove curative. It is extremely difficult to understand how it can cure simple hyperplasia if there is no dilatation, no catarrh, and a pylorus which permits the passage of a certain amount of food through it. On the other hand, it should prove beneficial when one or more of the secondary effects are present. Then, if the obstruction is not so severe as to be incompatible with life, the patient may recover, but the recovery is from the secondary effects and not from the hypertrophy. Such cases might, therefore, be regarded as unimportant if the child is not so fed as to develop these secondary effects.

Basing my opinion on the above arguments as to the pathology and mode of action of medical treatment, I hold that there is grave danger of ascribing to pyloric hyperplasia the symptoms due to spasm, with or without dilatation of the stomach; that there are mild degrees of hyperplasia compatible with life and giving rise to no serious symptoms; that some of these mild cases depend for their fatal issue on secondary complications which are curable by diet and lavage; that the majority of the cases tend to gradual contraction of the circular muscle and a degree of obstruction which is incompatible with life and only curable by surgical methods. And I protest most strongly against delay in adopting surgical measures, as I am convinced that the bulk of the supposed cures of this affection by medical means are really instances of simple pyloric spasm. Great care is essential in distinguishing between spasm and hypertrophy, otherwise it is undoubted that many unnecessary operations will be performed. (*Read January the* 17*th,* 1908.)

Discussion.

The Chairman (Mr. Sydney Stephenson) said he thought members would agree that it was the reading of papers such as those just submitted which justified the continuance and the prosperity of the Society, consisting as it did largely of specialists in the diseases of children. Questions of pathology, pathogenesis, and, above all, treatment had been touched on, and he hoped all those points would be fully and freely discussed. There seemed to be a conflict of opinion between the authors as to the value and the necessity of surgery in the condition.

Dr. G. A. Sutherland said he was glad to hear that Dr. George Carpenter had joined that small body of physicians who believed in the beneficial effects of medical treatment in those cases. Dr. Cautley seemed to be hovering on the brink, not having yet made the plunge. Evidently he did not yet think, with many, that definite typical cases of the affection had been cured by medical measures. He, Dr. Sutherland, protested against the assumption made by Dr. Cautley on the present and other occasions, that if pyloric stenosis were cured medically it must have been in such a mild form that it would have recovered without treatment. In his cases which were treated surgically, he had had the opportunity of studying the parts post-mortem. He admitted that Dr. Cautley had had a certain number of successful results from surgery, and he was naturally encouraged thereby. But the operation was a very serious one in young children, and Dr. Cautley had had the benefit of a surgeon who possessed special skill in that direction. Even then there was great danger to the life of the child, and many difficulties in connection with the treatment. Many infants had stood the operation, but had died afterwards from bowel trouble of some sort. So that the cases in which he would counsel resort to operation would be very few. Dr. Cautley said one ought not to delay, because the gravity of the operation was increased if the child were still further reduced. But he, Dr. Sutherland, would say that in all cases medical treatment should be given a trial first. Dr. Carpenter gave opium in his case, and it would be interesting to hear what was the result of that, and on what grounds that was based. He thought Dr. Carpenter accepted the view that there was considerable spasm about the pylorus, but as the hypertrophy was the chief underlying condition, he did not think that opium in sufficiently large doses could be safely given to an infant. Dr. Carpenter still thought that for a complete diagnosis one should be able to feel a pyloric tumour. He, Dr. Sutherland, did not think that was necessary before proceeding with the treatment. The pylorus was a very difficult thing to feel in an infant. Sometimes it could be felt, and sometimes not. One was entitled to diagnose the condition if the typical clinical symptoms were present—vomiting, constipation, wasting, and marked visible peristalsis. In some cases a slight peristaltic wave could be seen passing across the stomach region, but that was different from the strong wave which could be seen and felt in this condition. Also, why did Dr. Carpenter give olive oil injections? Were they to get fat into the system? One of the difficulties in those cases was to get fat into the system. He thought that where trouble arose when breast milk was given, the fault was not with the milk but with the manner of feeding. Dr. Carpenter said he gave five-ounce feeds to his child, which he, Dr. Sutherland, thought too large for such a case. Small feeds of the most digestible materials were required in such cases, and if there was a residue of food left in the stomach after washing out for a day or two, the infant was not having suitable food.

Mr. Lockhart Mummery said he had operated upon two cases of the condition, both of them Dr. Carpenter's patients. If Dr. Carpenter's view were accepted, it would mean that the poor surgeon would only get the cases of pyloric stenosis when they were absolutely hopeless; they had been nearly so before. He did not object to that, as he agreed with Dr. Sutherland that they were extremely unfortunate cases for operation; it was scarcely possible to have more unsuitable subjects for a severe operation. The infants were very young and badly nourished, and had probably been vomiting all the nourishment given to them for some days before. And the operation must either be pyloroplasty, stretching of the pylorus, or gastro-enterostomy, either of which was severe under any circumstances. Very few of the cases died on the table; but such an operation in such a patient must inevitably be fatal in a majority of the cases. The only patients which seemed hopeful for operation were those seen early, before there had been wasting, and in whom there was a fair amount of fat. Statistics would show that that was the kind of case which survived the operation. Those were also the cases in which, presumably, medical treatment would also have a good chance. He believed if medical treatment failed it would be almost useless to then hand the case over to the surgeons.

Dr. Charles W. Chapman said that in the condition under discussion there was a great deal of irritation causing the closure, and he was therefore surprised that no one had suggested the administration of carbonate of bismuth. If there was one thing which did good for little children with reflex irritation of the stomach it was bismuth, and it might be given fairly freely, even as much as small teaspoonful doses.

Mr. Hugh Lett said that the members of the Society were much indebted to the readers of the papers, as the subject was one of great importance. Surgical treatment was a very serious matter, and in many cases by the time that the surgeon was called in the condition was practically hopeless. The physician should be clear at the beginning of his treatment how long he would continue medical measures, lest by unduly prolonging them the prospects of operation were seriously affected. He advocated the claims of gastro-enterostomy as being simpler than pyloroplasty, and safer than Loreta's operation.

Dr. Reginald Miller said it was evident that it would be best to treat the condition on a pathological basis, as Dr. Cautley had tried to do, but it was doubtful if there were yet sufficient grounds to go upon. He, Dr. Miller, suggested that the symptoms of pyloric stenosis were entirely referable to spasm. He said that, firstly, because of the date of the onset of the symptoms. At the Clinical Society last year forty cases were collected, and in over 70 per cent. of them the onset did not occur until the third week of life or later. He had seen a case in which diarrhœa preceded the onset of the symptoms. It was true that many babies vomited from birth, but not sufficiently to cause wasting, and the well-marked constipation was frequently absent during the first two or three weeks of life. These facts proved that there was no marked blocking at the pylorus as a congenital lesion. Secondly, only the circular coat was hypertrophied. Thirdly, that if the baby did not live with lavage and careful dieting, it usually died of diarrhœa, showing that the pylorus was once again patent; presumably the spasm had been relaxed. A fourth and controversial point was that a few cases got well in which gastric peristalsis was observed week after week, and in which the tumour might be palpable for weeks. Such cases strongly simulated the real condition to which Dr. Cautley referred, and he, Dr. Miller, was convinced that they were quite identical. It was difficult to say what was the cause of the spasm. He had thought that it was due to a congenital error in the gastric juice, and he had tried to find what that defect was in the observations alluded to by Dr. Cautley. He believed that hyper-acidity was not the cause. Hyper-acidity was found in babies producing symptoms which ran a different course, generally getting well in a short time. Hyper-acidity associated with pyloric spasm without hypertrophy occurred in older and less wasted children: the tongue was clean, and there was no true peristalsis, although the viscus might stand up on palpation, and no tumour could be felt. He submitted that whatever might be the cause of the condition the symptoms were due to pyloric spasm, and that there was no doubt that this could be overcome by medical means. The ultimate result of the case depended upon how far the atrophy of the small intestine had proceeded.

Dr. George Carpenter, in reply, said he was in accord with Dr. Sutherland in regard to the uselessness of drugs for the condition; he had tried all sorts of anti-spasmodics in the affection and in cases of a functional nature, but none had done any good. Opium and cocaine had been valueless. In the case just recorded, he had only been responsible for the general principles of treatment, the treatment itself having been admirably carried out by Dr. Munro, to whom he desired to accord a full share of the credit of the case. He agreed that small feeds were better than large ones, but he was at issue with Dr. Sutherland in regard to diagnosis. In cases displaying symptoms of obstruction of the pylorus he thought there were two, if not three conditions; in one there was an underlying organic basis in the hypertrophied pylorus, and in the other there was no such abnormality, and they were, he thought, both attended by the same symptoms. Certainly violent expulsive vomiting and pronounced gastric peristalsis occurred, and other symptoms suggestive of congenital hypertropic stenosis of the pylorus arose in dyspeptic infants who were not suffering from that complaint; he had had numerous typical examples under his care which recovered, and which he was satisfied were not examples of congenital hypertrophic stenosis of the pylorus. But there was one distinction wanting in the spasmodic form, namely, the detection of a pyloric tumour, and that he looked upon as very important. Though it was difficult to find a pylorus by ordinary methods of abdominal examination, as it slipped away, yet if the stomach walls and underlying parts were pinched up between the thumb and finger, it was easy to grasp the small tumour. With regard to the pathological findings in those cases, he had examined three cases microscopically, and in all of them there was hypertrophy of all the muscular fibres. And in the cases he had seen the stomach walls, for a quarter of an inch from the pylorus, were hypertrophied also. The question was whether the stomach was hypertrophied simply because it had more work to do, or whether it was a congenital defect, like the condition of the pylorus. He thought it was due to congenital defect, and that in such cases there was an added condition, possibly spasm, but rather some other pathological finding, such as gastritis, perhaps a congested mucous membrane, which prevented the food going through the stenosed pylorus. He thought it was impossible to express any very positive opinion about the "added condition," but it was the "added condition" which was responsible for the symptoms, which were not solely due to the hypertrophied pylorus. Surgeons had had their innings, and had done their best, and physicians had relied upon them, but he did not think the surgeons had been as successful with those cases as could have been wished. It remained for physicians to see whether they could not do better. In any future cases of undoubted hypertrophic stenosis of the pylorus which came under his care he would certainly employ medical measures.

Dr. Cautley, in reply, said the matter was one of very grave difficulty and the utmost importance. The lives of many infants would be sacrificed before it was settled as to what was the right treatment. As Mr. Mummery said, if the baby was to be submitted to prolonged medical treatment before being handed over to the surgeon it would almost surely die. In hospital practice these babies were not brought until they were much wasted, and often the wasting continued while medical measures were being tried. He hoped he had made it clear that he was not now as dogmatic as he was some years ago on the necessity for surgical treatment. He was still convinced that there was a pure hyperplasia of the pylorus, which was not necessarily fatal by itself, though in most cases it tended to contraction and secondary effects which resulted fatally. These secondary effects depended upon the simple contraction of the hypertrophied muscle. His own cases, which he had referred to as dying under medical treatment, were characterised by moderate hypertrophy and obstruction, with no marked dilatation of the stomach or peristalsis. They died because there was not a sufficiently large lumen to the pylorus to allow enough food to pass through. At present he accepted the view that there was such a thing as pyloric spasm, which might be grafted on to hypertrophy and be a cause of the symptoms. Pyloric spasm might be easily mistaken for pyloric hypertrophy. It was remarkable in this connection that nearly everyone held strongly that drugs were useless, and he could not understand why that should be if the condition was due to pyloric spasm. Many of the children were not only breast fed, but fed in a proper manner. He recalled one case in particular where he was called in consultation to see a child when it was fourteen days old, because the obstetrician who attended the mother in her confinement suspected the condition was present, but he, Dr. Cautley, could not find sufficient evidence of it. The child was breast fed, and he arranged and gave directions exactly how the nursing was to be regulated. A fortnight later he saw the child, and concluded that the condition was present. The child was then fed at short intervals on whey and albumin water, with anti-spasmodic drugs, but nothing proved useful. As soon as an attempt was made to increase the feeds the child vomited. Such cases probably only surgery would benefit. He did not think the surgeon should be called immediately the child came under observation. None of the cases which he had referred to, with one exception, had been submitted to surgery until it had been under medical observation for at least one week.

Postscript to Dr. George Carpenter's communication, No. 38.

September the 6th, 1908.—Dr. Munro reports that the boy is now 17 months old and weighs 31 lbs. His mother, who has just returned with him from the seaside, says that he is "ripping," and "he walks and speaks a lot."

39. A CASE OF CONGENITAL HEART DISEASE.

By T. R. WHIPHAM, M.D.

The patient is a girl, aged 17 years, who is said to have been short of breath all her life, and to have been subject to attacks of vomiting on taking exercise. The feet are liable to swell, especially at night. There is no history of rheumatic fever, but three years ago the patient suffered for a time from "rheumatism in the feet." She has never had chorea nor any of the specific fevers common to childhood. There are three other children in the family, all of whom have good health.

The patient is a well-grown girl, whose cheeks are red, but not definitely cyanosed. The facial coloration is a family characteristic. There is no clubbing of the fingers or toes and the nails are of a good colour. The left side of the heart is considerably enlarged, the apex-beat being in the sixth space considerably outside the nipple line and somewhat forcible in character. Over the upper part of the sternum at the level of the second costo-chondral articulation there is a marked thrill. The percussion dulness extends on the left from the second rib to well outside the nipple and downwards to the apex, but on the right does not reach the right border of the sternum. Over the area of the thrill and in the second interspace to the right of the sternum a loud, rough systolic murmur can be heard, replacing the first sound. The murmur is audible all over the præcordium, but is most intense at the base and to the right. At the apex a first sound can be heard preceding the murmur. A blood count showed no polycythæmia.

Considering the age of the patient, the absence of cyanosis and of clubbing, the position of the thrill and maximum intensity of the murmur, and of the obvious hypertrophy of the left ventricle, I think that the lesion in this case is not a pulmonary one, but some abnormality of the great vessels. Possibly a patent ductus arteriosus is present. (*Exhibited January the 17th*, 1908.)

Discussion.

Dr. Charles W. Chapman said he thought there was aortic stenosis; the well-marked thrill and the rough character of the sound indicated considerable stenosis. But the girl's fingers were not clubbed, and there was no cyanosis. She was subject to faintness and giddiness, from a deficient blood supply to the brain. He did not think it was a case of patent ductus arteriosus. A girl, aged 18 years, was brought to him, supposed to have aneurysm, and there was the same condition—an intensely roughened first sound. It was certainly a congenital case.

Dr. T. R. Whipham, in reply, said there were some points against the supposition that it was a case of aortic stenosis. The cardiac beat was quicker and rather more forcible and sudden than was usually met with in aortic stenosis. He was inclined to think the aorta was free, but he was obliged for the suggestion.

40. CASE OF SYPHILITIC SYNOVITIS.

By J. PORTER PARKINSON, M.D.

John T—, aged 7 years, had a marked family history of inherited syphilis. His mother had nine miscarriages, two out of six children born alive, four died at, or shortly after, birth, and the other surviving child has been under the charge of Mr. Sydney Stephenson for interstitial keratitis.

This patient was always weakly and had suffered from measles, whoopingcough, chickenpox, etc. The left eye was diseased at 2½ years of age, and a large leucoma of the cornea is the present result.

Towards the end of October, 1907, he began to suffer from pain in the legs, and early in November he came to the out-patient department of the Queen's Hospital for Children for this and was found to have periostitis over the upper third of both tibiæ, and shortly afterwards was admitted as an in-patient, as while under mercurial treatment synovitis had appeared in both knee-joints.

He was a fairly well nourished boy, of a syphilitic aspect; the upper central incisors, recently cut, were separated and pegged, but not notched. There was no scarring of the mucous membrane of the mouth. There was no evidence of disease of the respiratory or abdominal organs. The heart was not enlarged, but there was a soft systolic murmur heard at the apex, conducted outwards for an inch or two, but no other evidence of heart disease.

There was thickening over the upper parts of both tibiæ, but the pain and tenderness had disappeared.

Both knee-joints were distended with fluid, but they were not tender nor painful and there was no extra-capsular effusion. The temperature was normal. The urine for the first four days in the hospital contained a trace of albumin and a few hyaline and granular casts, but this all cleared up and did not reappear. A skiagram showed that the bony structures of the joint had a normal appearance, though there was slight thickening of the upper parts of the tibiæ, the seat of the previous periostitis.

Scott's dressing was applied to the joints, and they were strapped, while the mercurial treatment was continued; this resulted in a very considerable diminution in the effusion after a week or ten days.

(*Exhibited January the 17th,* 1908.)

Discussion.

The Chairman (Mr. Sydney Stephenson) said he recognised in Dr. Porter Parkinson's patient a child who was under his own care some years ago with a large leucoma covering the greater part of one cornea. It was not in the nature of interstitial keratitis, but followed a septic ulceration of the cornea. He did not see it in the acute stage. He remembered doing an iridectomy downwards and inwards to improve vision. He thought the way the mercury was used had something to do with the result. Some of the syphilitic joints associated with interstitial keratitis yielded to intra-muscular injections of mercury. In trying to get rid of those effusions he had used blisters, and it was interesting to note that the marks left by them had become pigmented.

Dr. F. Parkes Weber said he was interested to hear that the case had been examined by Röntgen rays, because he believed that in syphilitic joint disease the rays were of great use in diagnosis. In two recent cases in adults of chronic swelling about joints, in which the diagnosis of tertiary syphilis was confirmed by the results of anti-syphilitic treatment, the rays showed much greater changes than would have been suspected from mere inspection. In one case the Röntgen photographs evidenced great thickening of the periosteum, and in both cases they proved that there were marked changes, probably of gummatous nature, in the bony surface of the affected parts.

Dr. G. A. Sutherland said he did not think that, as a rule, they were difficult and obscure cases, such as Dr. Parkes Weber referred to. Dr. Parkinson's case was a typical one of painless synovial effusion in a child at the usual age. It was contrary to his (Dr. Sutherland's) experience to find rapid improvement under mercurial treatment. In his experience the fluid came and went, not under the influence of mercury, and recurrences were very common. He had seen cases which had been operated upon at general hospitals, and yet the condition had recurred. It seemed to be more like the associated condition of interstitial keratitis, so far as the results of mercurial treatment showed.

Mr. Hugh Lett considered it a typical case of synovitis of the knee due to inherited syphilis, and he thought these cases were important as they were occasionally diagnosed as being due to some other cause. He had seen several cases which had been treated with splinting for some time in consequence of a diagnosis of traumatic synovitis. In another important variety of the congenital syphilitic joint there was considerable thickening of the synovial membrane, with little or no effusion into the joint. Both of these types, viz. with or without effusion, were occasionally treated as tuberculous joints. The most striking feature in these cases was the absence of pain on movement.

Dr. Porter Parkinson, in reply, said he stated there were no bony outgrowths visible by X rays in or near the joints, but there was evidence of thickening below, which he thought to be due to the periostitis, which could be felt. The child had been under treatment for a month only, so it was too early yet to speak of cure. The effusion had not disappeared, and the effusion in the knee-joint appeared while the child was under mercurial treatment for periostitis of the tibia. He thought that cases which were chiefly hydrarthrosis—whether due to syphilis or other conditions—were specially resistant to any general treatment. Where Scott's dressing was beneficial he thought it was due to the pressure it exerted. In most such cases he did not think there could be much difficulty in the diagnosis, but he recently read a paper contributed to a French journal stating that many mistakes had occurred between that condition and tubercular joint disease, children having been put up in splints and plaster for months under the impression that the disease was tubercular.

41. CASE OF CRETINISM.

By G. A. SUTHERLAND, M.D.

A. C—, male, aged 9 years. The patient seemed normal during the period of lactation, ten months, but afterwards his rate of growth became very slow. He was a quiet, well-behaved child, who did not take much interest in other children. He went to school at the age of seven years and made slow progress. At the age of five treatment by thyroid extract was begun at a hospital and continued for two and a half years, when the doctor stopped it, and said it was only to be resumed if the patient became worse. (This statement is open to grave doubt.) During the treatment his rate of growth improved and he became brighter.

On admission into hospital his height was 36½ inches and his weight 33 lbs. 13 oz., and his appearance suggested an age of about three years. He was a pale, languid, stout-looking child, with the general characteristics of cretinism. He was extremely sensitive to cold. Placed in a chair in front of the fire he would remain placidly content for an indefinite period. There was an appearance of abnormal muscular development about the limbs, the result probably of the defective growth of the long bones. Under treatment by thyroid extract (1 gr. twice daily) rapid improvement took place. Active voluntary movements took place, and he joined in the play and talk of other children. Treatment by pituitary extract had been combined with the thyroid in order, if possible, to induce more active growth in the long bones. For five weeks he took 2 gr. of pituitary extract twice daily. There had been no perceptible effect either on the growth of the bones or in the production of diuresis.

(*Exhibited January the 17th*, 1908.)

Discussion.

Dr. F. Parkes Weber said he had never seen or heard of any improvement in any condition from pituitary gland treatment, that is to say, in any case coming under his own observation. He did not remember having ever used the treatment himself.

42. CASE OF TUBERCULOUS PERITONITIS.

By G. A. SUTHERLAND, M.D.

A. L. W—, female, aged 9 years. This child was admitted into hospital in August, 1907, with a history of five months' illness characterised by diarrhœa, pain in the abdomen and wasting. At times blood had been present in the motions. She had been breast fed for seven months and afterwards on cow's milk. At the age of four years she had suffered from whooping-cough and measles. One other child in the family of twelve had died of some form of tuberculosis.

Her condition on admission was a very serious one. The child was pale and wasted. At night she was often delirious and during the day she had hallucinations. There was constant twitching about the hands and fingers. She lay in a stuporous condition. There was incontinence of urine and faeces. The abdomen was distended and hard, resonant all over, and deep palpation was impossible, but no masses of any kind could be felt. The motions were loose, yellow and offensive. Calmette's ophthalmic test gave a very marked reaction. There was no definite evidence of tuberculosis apart from the abdomen. The temperature was raised at first, between 99° and 100° F. at night, but later became subnormal. As her appetite was good she was allowed full diet, practically what she liked, as the case appeared for some time to have passed beyond the stage of curative treatment. About three months after admission improvement began. Her mind became calmer, her interest in things about her increased, the motions were healthier in character and the incontinence ceased.

The case is shown as an example of the wonderful recovery which may take place even in the severest forms of tuberculous peritonitis. This was one of the most hopeless cases the writer has seen. It illustrates the fact that in the absence of general infection and local complications (pus formation, ulceration of the bowel, etc.) the ultimate prognosis in tuberculous peritonitis is good, although the course of the disease may be a prolonged one. At the end of five months this child may now be described as being convalescent, although the abdomen is still distended, and her physical powers are weak. She has been in the country, and has undoubtedly benefited much from the fresh air treatment there and on the hospital balcony.

(*Exhibited January the* 17*th*, 1908.)

Discussion.

Dr. Edmund Cautley said he agreed with Dr. Sutherland that the prognosis in uncomplicated tuberculous peritonitis was eminently favourable if there were no tuberculous masses and no purulent effusion. These were cases of adhesive peritonitis, or those in which serous fluid was effused, even in considerable quantity. If such patients died, it was found that they had secondary complications, generally tuberculous disease in the lungs. Persistent treatment by diet and drugs was essential. He asked whether drugs were used in the case.

Dr. George Carpenter said he had followed up a series of cases of tuberculous peritonitis which he had published in 'International Clinics,'* and he was surprised at the number of deaths. Thus of 31 children 15 died, 6 were doubtful, and 10 were alive at the time of writing, and so far as was known in fair health. He had then stated that tuberculous peritonitis is not infrequently cured, and perhaps more often resolves than any other form of tuberculosis, but that he was not prepared to think it other than a very fatal disease. Since writing this he had seen no need to alter his views. In the special discussion on tuberculous peritonitis † he had pointed out that "implication of other organs in fatal cases of primary abdominal tuberculosis is not uncommon, and of all organs and structures the lungs appear to be most likely to be attacked. Invasion of the lungs in such cases probably occurs *viâ* the thoracic duct and pulmonary artery, the lungs acting as a filter. This is contrary to the experience of others who consider that food tuberculosis may get well or spread, but never leads to tuberculosis of the lungs." Sometimes these cases die of meningitis.

Dr. G. A. Sutherland, in reply, agreed that many such cases died, and that they did so from some such complications as had been mentioned. When the disease was generalised the cases were invariably fatal. The child had no special diet at the time; she was practically moribund, and so she had what she liked. When a gradual improvement was noticed he put her on a protein and non-fermentative diet, and under that the bowel condition improved rapidly, the abdominal distension went down, and she steadily got better. A combination of open-air treatment and diet brought about the cure; drugs had no part in it.

* Vol. iii, p. 83, 1891.

† 'Reports,' vol iii, p. 109, "Tuberculous Peritonitis."

43. NOTES ON A CASE OF POLIO-ENCEPHALITIS.

By LEONARD GUTHRIE, M.B.

The patient first came to hospital a year ago because of a nasal intonation in her speech. She was found to be suffering from enlarged tonsils and adenoids, and these were accordingly removed, ethyl chloride being administered. She had been perfectly healthy until the operation, but vomited after food for the following two weeks and then developed measles. The attack was of the usual severity and was not peculiar in any way. When convalescent it was found that her lower extremities were weak, for she fell down frequently, and if she walked any distance she became very tired and fell more readily. The upper extremities were also affected; she has not been able to feed herself since the attack. There was frequent headache and pain in the left ear and the extremities of the left side pained her irregularly.

On admission she was found to be an intelligent, healthy, and well-coloured plump girl, aged 5 years. There was no squint and no interference with the muscles of the face or tongue, no spasticity, or wasting of muscles. The knee-jerks were much exaggerated and ankle clonus well marked on both sides. The ankle clonus was peculiar in that its rate was rapid (336 per minute), and the only superficial reflex that could be elicited was the left epigastric. Sensation was not affected. Heat and cold were confused on the outer aspect of both legs, not elsewhere.

There was inco-ordination in movement of the extremities and a tremor accompanied these movements. There was incontinence of urine and fæces. No reaction of degeneration.

When attempting to sit up, the upper part of the body fell forward and the head fell forward on the chest. She was unable to stand or walk without assistance. The gait was peculiar, cross-legged in character. The knee was flexed, the foot lifted high and carried forward, and then by adductor spasm the foot was brought across in front of the stationary foot and rotated so that the toes pointed inwards. The ankle became extended and rigid when attempting walking, so that she walked on her toes.

Special senses.—Nothing to note; child is otherwise healthy.

There have been no previous illnesses and there is no family history of tuberculosis or syphilis.

This case is interesting in that it simulates a condition of congenital spastic diplegia, but is an acute polio-encephalitis, and the lesion is probably to be located in the motor areas of the cortex.

Since being in hospital the patient has improved, and is now able to hold her head up for a short time and to sit more upright. The left hand, too, is becoming more useful. The prognosis, however, is not hopeful, the improvement being probably due to an improvement in the general condition of the patient as the result of hospital life. (*Exhibited January the 17th,* 1908.)

Discussion.

Dr. F. Parkes Weber pointed out that the cerebral diplegia in this case was not congenital, as it usually was, but followed a definite febrile illness—in this instance, measles. He believed that occasionally cerebral diplegia was acquired at a later age than in Dr. Guthrie's case. Several years ago (1892) Sir Dyce Duckworth and Dr. H. H. Tooth published a paper on "Two Cases of General Spastic Rigidity," in vol. xxviii of the 'St. Bartholomew's Hospital Reports,' page 263. He saw one of them, a girl about thirteen years old, who had a severe febrile illness, of uncertain nature, accompanied by symptoms of pseudo-bulbar paralysis as well as by spasticity of all four limbs. He believed now that the patient in question may have been affected by bilateral polio-encephalitis, possibly associated with a certain amount of basal meningitis. He thought all such cases were of extreme interest, and that they should all be carefully recorded.

44. A CASE OF SUPERNUMERARY AURICLES IN AN INFANT.

By P. LOCKHART MUMMERY, B.C.

The patient is a boy, aged 3 months, the youngest of six children, the others being quite free from congenital defects. The child is a healthy infant. On both sides of the head there is a supernumerary auricle situated just in front of the external auditory meatus, in the position of the intermaxillary cleft. The extra auricle on the right side is about half the size of the normal ear, that on the left side is much smaller. The external ears are on both sides quite normal. The child has a coloboma in the left eye. There are no other congenital defects.

(*Exhibited January the 17th*, 1908.)

45. A CASE OF INFECTIVE ENDOCARDITIS; IMPLICATION OF AORTIC AND MITRAL VALVES.

By GEORGE CARPENTER, M.D.

Annie R—, aged $10\frac{1}{2}$ years, came under observation on October the 3rd, 1907. She had been ill four weeks, but before that had appeared to be in good health. She had had measles and whooping-cough. Twelve months previous to the present illness she had "bad pains in her legs and could not stand for about a fortnight, being in bed for a week of that time. Then she began to waste, was drowsy," and, as her mother remarked, "sat about." For the last week "she had been very short of breath and unable to walk, and complained of pain in the left side." The child's sister who slept with her stated that the night before she was brought to the hospital "her heart made a noise just as though it were whistling" and it was "beating very hard." She had not noticed anything amiss with it before then.

There was no family history of rheumatism. Her mother was healthy. Her father had died of consumption.

State on admission.—A delicate-looking but not pallid child with fair hair, dark eyelashes, and pulsating arteries in her neck. Pulse-rate 126 to the minute and the pulse is perhaps a trifle collapsible, but it is certainly not typical of aortic regurgitation; respiration-rate 24 and temperature 98° F.

Cardiac impulse.—The heart is seen beating in the fifth and sixth interspaces and 1 inch outside the nipple line; the impulse is accompanied by a systolic thrill.

Area of dulness.—The area of superficial cardiac dulness commences at the third left costal cartilage and does not extent beyond the mid-sternum.

Sounds.—*At the base* are systolic and diastolic bruits, heard equally well over the aortic and pulmonic orifices. The systolic bruit is conducted in to the carotid arteries. The diastolic bruit is heard in the usual situation of aortic regurgitation, viz. down the left side of the sternum.

At the apex there is a pre-systolic bruit—this is not specially characteristic, but it is pre-systolic in point of time and is rather rumbling in character. There is also a loud systolic bruit which is conducted into the axilla. The systolic bruit at the base is quite different in character from that at the apex.

Liver.—This percusses two and a half fingers' below the costal margin.

Spleen.—This is enlarged; its free edge is a trifle over 3 inches from the umbilicus; it is notched but not tender.

Urine.—Free from albumin.

Eyes.—No change in the fundus oculi.

November the 18th.—She has now been in hospital for a little over six weeks. During the first month of that time the temperature was for the most part sub-normal; twice only was it raised, and on both occasions to less than 100° F. Since then it has been rather more undulatory, but even then the highest record was only 100·2° F. The pulse-rate has varied between 100 and 128 to the minute, the respiration-rate between 24 and 40—the average about 26. The daily quantity of urine has varied between 13 and 31 oz.; it has been free from albumin and blood, and twice was centrifuged for the detection of red blood-corpuscles with negative results. She gained ½ lb. in weight after a month's residence in the hospital. Her bowels were opened from one to three times a day. She had been "much more comfortable and slept well at night."

But on this date her physical condition is as follows: Her face is puffy, the abdomen is swollen, there is a fluid thrill and shifting dulness. The liver is fully three fingers' breadth below the costal margin and the spleen is felt to the same extent.

Heart.—The apex beat is in the sixth interspace ¾ths inch outside the nipple line. There is no extension of cardiac dulness to the right in spite of the fact that the liver is larger and more prominent than before.

Over the apex there is a pre-systolic "rumbling" bruit followed by a blowing mitral systolic murmur, which is conducted well into the axilla and is (?) heard faintly in the back.

Over the base there is a "to-and-fro" bruit which is now best heard over the *aortic area.* Down the left side of the sternum the diastolic bruit is typical in character, though its special regurgitant distinctive whiff is not audible at the base. The pulse on critical examination does seem to collapse a trifle, but it is certainly not characteristic of aortic regurgitation. No swelling of the feet. No albumin in the urine.

November the 25th.—She is not so well to-day. The face is slightly puffy, the lips are pale, and the conjunctivæ rather jaundiced. The hands are swollen and there is œdema of the feet and legs, and the skin over the chest and abdomen is readily marked by the clothing. The liver dulness now extends almost to the umbilicus, where its free edge can be felt. But the area of cardiac dulness has not increased, though the heart's action is more tumultuous (140 to the minute), and the respirations are 40 to the minute (much increased). There is a little albumin in the urine (it was absent on the 20th inst.), but no microscopical evidence of blood.

At 5 p.m. the house-physician was called to the ward and found her *in extremis*, suffering great pain at the epigastrium and pulseless at the wrist. In spite of treatment (hypodermic injections of strychnine, and brandy, and oxygen inhalation) she died fifteen minutes after his arrival.

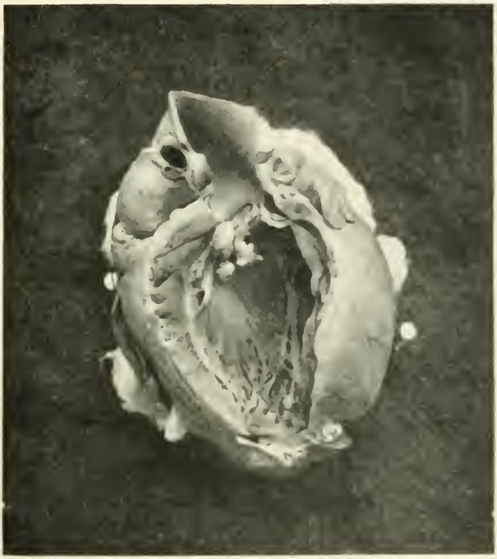

FIG. 41.—Fungating vegetations on the aortic valves. (From a photograph by Dr. A. W. Harvey.)

Autopsy.—November the 26th—twenty-four hours after death. Rigor mortis not present. The abdominal cavity contained a pint of fluid; there were a few ounces of fluid in each pleural cavity, which showed a few old adhesions on the left side and some recent pleurisy at both apices.

The lungs were œdematous; the bronchial glands enlarged and fleshy.

The pericardium contained a small amount of fluid, but there were no adhesions.

Heart.—The left auricle and ventricle were both dilated, and the left ventricle was also a little hypertrophied.

The right auricle and ventricle were somewhat dilated, but the heart had not lost its shape and there was no rounding of the organ in this direction.

The aorta was dilated. Masses of fungating vegetations were seen on the aortic valve hanging into the cavity of the ventricle. Half an inch above the valve on the aorta itself was a vegetation.

The mitral valve displayed a row of pink granulations on its ventricular aspect; it admitted two fingers.

The tricuspid valve admitted three fingers, and it and the *pulmonary valves* were healthy.

The liver, which was much enlarged, was early nutmeg; it showed some pale areas, and if anything was a trifle greasy on section. Weight, 32 oz.

The spleen was friable and coarsely granular, the grains being about the size of No. 5 shot. It was much enlarged, weighed 10 oz., and was adherent to the surrounding structures. There was an accessory spleen. No infarcts.

Stomach.—Rather large, but apparently normal.

Intestines.—Normal.

Genito-urinary tract.—Normal. Kidneys rather tough. Brain not examined.

(*Exhibited January the 17th,* 1908.)

46. A CASE OF ACUTE INTESTINAL OBSTRUCTION PRODUCED BY A BAND, THE LATTER BEING, IN PART, AN INTESTINAL DIVERTICULUM BEARING AN ACCESSORY PANCREAS.

By H. S. CLOGG, M.S.

An accessory pancreas is by no means a common abnormality, and when present it has been found in the wall of the stomach or small intestine, not very far distant from the main gland. Such accessory glands are purely of anatomical interest. But when situated in an intestinal diverticulum it may have also some considerable surgical importance. Looking over the literature it seems that an accessory pancreas in connection with an intestinal diverticulum is a condition of extreme rarity, and apparently there may be two distinct anatomical types. The one variety is that in which the accessory gland is developed in connection with a true congenital intestinal diverticulum; the second, in which the accessory gland has produced a false traction diverticulum. Zenker (1) has reported a case showing a true diverticulum situated 54 cm. above the ileo-cæcal valve; the diverticulum was 5 cm. in length, and was provided with a mesentery rich in fat. In this mesentery, close to the end of the diverticulum, was an accessory pancreas. Zenker considered this diverticulum as a true one—a Meckel's diverticulum. Schirmer (2) records an instance where a diverticulum was situated over 1 m. above the ileo-cæcal valve; it was 8 cm. in length and was provided with a small mesentery. At the extremity of this diverticulum was a small accessory pancreas. The length of the process and the presence of a mesentery suggest, in this case, that it was an instance of a true congenital diverticulum. Brunner (3) records the case of a child, aged 4 years, who was suffering from intestinal obstruction. Operation revealed an intussusception of the small intestine, and the apex of the invagination had traversed the ileo-cæcal valve. The commencement of the intussusception was an intestinal diverticulum which was situated about 37 cm. above the ileo-cæcal valve. Brunner regarded this as a true Meckel's diverticulum. Attached to the diverticulum was an accessory pancreas. Bize (4) relates the case of a child, aged 3 years, who suffered from an intussusception. At operation the intussusception was reduced, but the child did not recover. About 25 cm. above the ileo-cæcal valve was a diverticulum, well developed with muscular and mucous walls, and at the end of this was an accessory pancreas. The diverticulum did not play any part in the intussusception. At the site of attachment of the pancreas the mucosa and the submucous layer cover it on the intestinal aspect completely; the muscular layer, however, is interrupted, and its fasciculi pass in between the lobules of the gland, and the peritoneum is continued on its exterior. Bize records a second case of a child, aged 6 years, upon whom an operation for intussusception was performed. The origin of the intussusception was a diverticulum which was situated about 60 cm. above the ileo-cæcal valve. At the base of the diverticulum was an accessory pancreas, conical in form with its summit directed towards the cavity of the diverticulum. The gland was situated partly in the submucous and partly in the muscular tissue. A further point of interest in this case is that portions of the gland were the seat of new formations, presenting the characters of true adenomata.

Neumann (5) found at an autopsy on a child, aged 10 months, about two feet above the ileo-cæcal valve a diverticulum, and fixed to the apex of this by a short pedicle was a small, solid, glandular body. The latter proved on microscopical examination to be an accessory pancreas, with an excretory duct, into which a probe could be passed. The accessory gland was enveloped in a muscular covering. Neumann inclined to the hypothesis that the diverticulum had been formed independently of the omphalo-mesenteric duct, and it had been produced little by little by traction of the accessory gland on the walls of the intestine.

Nauwerke (6) found in the ileum two diverticula. The one was situated 2 m. above the cæcum, and it bore an accessory pancreas with an excretory duct. The second diverticulum was situated 80 cm. above the valve. Nauwerke considered it certain that the second diverticulum was the true diverticulum of Meckel, and the first which had the accessory pancreas was a secondary development produced by traction.

The case which I report is, I think, an example of an accessory pancreas in a congenital diverticulum.

The notes of the case are as follows :

E. T—, aged 12 years. Previous to this illness he had always been a healthy lad, and had never suffered, so we were told, from any abdominal trouble. On July the 17th, 1907, when playing with his schoolfellows on his way home from school he received a blow in the abdomen. This caused him some pain, but did not produce any collapse at the time. He walked home alone, a distance of half a mile, but during the walk he vomited five times. On arriving home he complained of great pain in the abdomen, he refused food and went to bed. During the night he had practically no sleep; the pain increased in severity, became distinctly paroxysmal, and he vomited several times. The pain at first was referred to the whole abdomen with maximum intensity at the umbilicus, but after a few hours he always pointed to the right iliac fossa as the site of maximum pain. Not until twenty-four hours after the onset of the illness was a doctor summoned. The boy then appeared extremely ill, with a subnormal temperature and a pulse of 120; vomiting was incessant, and the vomit was bile-stained. The abdomen moved freely on respiration; it was not generally distended, but some fulness was noted in the right iliac fossa, and the boy referred all his pain to this and the hypogastric region of the abdomen. The pain was distinctly paroxysmal in character, and in between these paroxysms of pain he was fairly comfortable. The only region of the abdomen which, on palpation, revealed any tenderness was the right iliac fossa. Later in the day, although no morphia had been given, the condition improved somewhat, the pain became easier and the vomiting ceased. The following morning, about forty-four hours after the commencement of the illness, a sudden change for the worse was apparent. The pulse increased in frequency, vomiting set in again, and the vomit was very copious and had a foul odour. The boy then looked extremely ill, his face was very pale and his aspect was most distressing. Pulse 140, but varied with the pain. He was constantly crying out with pain, which he located chiefly to the right iliac region. The pain was paroxysmal, but the paroxysms recurred at very short intervals. The abdomen was only slightly, if at all, generally distended, but in the right iliac fossa was an obvious fulness, and if examined in the interval of freedom from pain, a well-defined tender, rounded, elastic, and resonant-to-percussion swelling was felt in this and the hypogastric area. This was just reached from the rectum. Gentle rubbing of the abdominal wall made no difference to the consistency of this swelling. Save over the area of the abdominal wall corresponding to this swelling there was no tenderness of the abdomen. The bowels had not been opened throughout the illness, and only once, as far as could be ascertained, had flatus been passed, and then after a rectal examination. The temperature had been subnormal throughout.

Operation about forty-six hours after the commencement of the illness. The peritoneal cavity contained a quantity of bloody fluid—at least a pint. The tumour previously felt was a distended coil of small intestine. This was jet black in colour, it had lost its lustre and was in places coated with inflammatory lymph. This coil of intestine, measuring about 10–12 inches in length, was snared by a band. The latter was attached to the mesentery in the neighbourhood of its termination in the right iliac fossa. After severing this band close to its attachment it was seen that in addition to being tightly snared the bowel had also undergone a rotation of 180° around its mesenterial axis. The band having been divided the volvulus was easily untwisted, but the bowel did not regain any lustre, neither was any peristalsis nor circulation observed in it. Deeming, therefore, it was in all probability gangrenous, it was excised. The band was seen to arise from the intestine immediately adjacent to the snared coil; it was ligatured and removed. The portion of bowel removed, measuring about one foot or more in length, included the site of attachment of the band. For various reasons the ends of the gut were united by means of a Murphy button, outside which a continuous suture was run and omentum wrapped around and kept *in situ* by a few sutures. A drainage-tube was inserted in the pelvis and the remainder of the wound was closed.

There was considerable collapse following the operation and for some three days afterwards. Strychnine and digitalin were given regularly hypodermically, and also subcutaneous infusions of saline, with rectal injections of saline and brandy. Vomiting commenced twenty-four hours after the operation and was very troublesome for two days. A powder of cerium oxalate and bismuth had more effect on this than any other remedy which was tried. Rectal feeding had at one time to be resorted to entirely. On the fifth day improvement commenced and vomiting ceased. From that time onward everything progressed satisfactorily and the boy made a speedy recovery. There was never any distension and practically no tenderness of the abdomen; occasionally during the first few days some complaint was made of colicky pains. For a few days some offensive fluid escaped from the tube; this soon ceased and the tube was left out at the end of a week. The button was passed on the ninth day.

Description of the strangulating agent.—This consists of three elements, viz. an *intestinal diverticulum*, a *cord* leading from this, and an *accessory pancreas*.

The diverticulum measures some ¾ths inch in length; it was ligatured before division, and hence the furrow around it close to its proximal end and the apparent inequality of its lumen in transverse diameters. To what part of the small intestine it was attached I am unable to state. The wall of the diverticulum is composed of mucous, submucous and muscular layers covered by peritoneum. At the distal end of the diverticulum is a flattened glandular mass of tissue measuring some ¾ths inch in diameter. This shows the structure of pancreas. This accessory pancreas projects slightly into the lumen of the diverticulum, and over this projecting or free border of the gland intestinal wall can be traced, including mucous membrane and some muscular fibre. The intestinal wall can also be traced some distance over the outer aspect of the gland. The intestinal wall differs in thickness in different places due to the degeneration of the mucosa and also to some thinning of the muscular layer. The pancreas seems to be developed directly in the wall of the diverticulum and is without any excretory duct. The cord is attached practically to the junction of the diverticulum and pancreas; it is a definitely rounded cord composed of fibrous and fatty tissue some two inches or more in length.

Whether this be an example of a true congenital diverticulum bearing an accessory pancreas or whether the accessory gland was developed in the wall of the intestine and by traction produced a false diverticulum I do not think it can be positively stated. I incline to the former view, and my reasons are: (1) The more or less complete structure of the wall of the diverticulum; and (2) the presence of a definite band or cord attached to practically the extremity of the diverticulum, having a similar attachment, therefore, as the omphalo-mesenteric cord has to a Meckel's diverticulum. It seems hardly possible that this band, attached as it is, can be the result of an adhesion.

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(2) SCHIRMER.—Quoted by Bize.
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(4) BIZE.—'Revue d'orthopédie,' 1904, p. 149.
(5) NEUMANN.—'Archiv des Heilkunde,' xi, 1870.
(6) NAUWERKE.—'Patholog. Anatomisch Mitteilung.,' 1892, xiv.

(*Read February the 21st,* 1908.)

Discussion.

Mr. Hugh Lett agreed that the specimen was a Meckel's diverticulum, and thought that the strongest argument in favour of this was the thick round cord which was connected with the diverticulum. The boy had never had any serious abdominal illness, and even if he had it was most improbable that it could have produced an adhesion of such thickness. It was also significant that this cord extended from the apex of the diverticulum. Barnard found that Meckel's diverticulum was responsible for acute intestinal obstruction twenty-one times in 669 cases, and the specimen shown by Mr. Clogg was an example of the variety which most frequently caused obstruction, namely, a diverticulum leading from the bowel, continued at its apex into a thick rounded cord, whose distal end, though originally free, had acquired a new attachment, and so formed an archway under which a loop of intestine passed and became strangulated. In the great majority of cases the cord was attached to the root of the mesentery, though it might be connected with various other structures, such as the small intestine, cæcum, meso-rectum, meso-colon, etc. Not infrequently the cord was attached to the umbilicus, but when that occurred the connection was probably always congenital, and due to the fact that the remains of the vitello-intestinal duct, which were represented by Meckel's diverticulum, had never become separated from the umbilicus. The prognosis in cases of acute intestinal obstruction due to Meckel's diverticulum was very grave, for of the twenty-one cases mentioned above, no fewer than sixteen died; that was a mortality of 76·2 per cent. compared with a mortality of 53 per cent. for all cases of acute intestinal obstruction. One reason which had been given for this high mortality was the frequency with which the diverticulum itself became gangrenous, in consequence of its being rotated on its own axis, and so strangulated by the coil of intestine which it obstructed. Mr. Lett heartily congratulated Mr. Clogg on the specimen he had brought before the Society, and on his most successful operation.

Mr. H. S. Clogg, in reply, said the distal end of the band was attached in the right iliac fossa, *i. e.* to the peritoneum on the posterior wall.

47. TWO CASES OF CONGENITAL HEART DISEASE.

BY GEORGE CARPENTER, M.D.

CASE 1.—*Aorta arising from both ventricles; small pulmonary artery with two semilunar valves; patent ductus arteriosus; semilunar valve efficiently guarding foramen ovale, but displaying a tiny perforation.*

Olive May F—, aged 10 weeks, was admitted into The Queen's Hospital under my care on December the 21st, 1907. Her parents were healthy. The mother had had twelve children and four had died. Of the latter two died from whooping-cough (twins), one, a full-time child, was stillborn, and one, a six-months premature child, was alive at birth and weighed only 1 lb. 2 oz.

The patient was "born with jaundice," but was a fair-sized baby; since her birth she had wasted. When two weeks old she was brought to the hospital, and had been a regular attendant since. She took food well and slept well, but was restless when awake.

When admitted into hospital she was found to be an anaemic, ill-nourished, and ill-developed child. The frontal and sagittal sutures were patent and the anterior and posterior fontanelles widely open; no craniotabes. The cartilages of five or six ribs were very ill-developed, and of the last four were certainly absent. Her tongue was large and swollen and protruded from her mouth, and she was apparently unable to close the lips.

Heart.—The first cardiac sound was best heard in the fourth interspace internal to the nipple line. The area of dulness was increased to the right of the right sternal margin. There was a loud systolic murmur best heard over the pulmonary orifice. It was audible over the chest, front and back, and heard better on the left side in front than on the right. The liver was not felt, the spleen was palpable. On January the 1st she had clonic convulsions on the left side during the afternoon, and was given a dose of syrup of chloral (♏v), when the fit ceased. During the night the pulse was very weak, she was much collapsed, and was given a mustard bath, and at midnight she was infused into the axilla with two ounces of saline and half a drachm of brandy. Death took place the following morning. While in hospital the temperature was raised on two occasions, once to 100° F. and once to 100·4° F.; it was otherwise normal or subnormal. She was never cyanosed at any time.

Post-mortem.—The aorta was situated over the right and left ventricles, four fifths of its lumen being over the latter.

Its cut end measured $\frac{1}{4}$th inch across and it had three normal semilunar valves.

The coronary arteries were normal.

Below the channel of the aorta was the upper border of a thick fleshy partition, the top of a septum ventriculorum. This border was rounded and lay underneath and close to the channel of the aorta. It took tendinous origin in front between the left and anterior semilunar aortic valves at the undefended space, and was attached about $\frac{1}{8}$th inch below their point of union. Behind it ended in a fibrous attachment, which joined the auricular septum, and which separated the mitral and tricuspid valves and was placed $\frac{1}{4}$th inch below the convexity of the left semilunar cusp near its junction with the posterior semilunar. The attached portion of the anterior cusp was hidden by this muscular bridge which stretched across it (*vide* Plate IV, fig. 1; the muscular bridge has been cut across and turned to the right).

The pulmonary artery was obviously small and measured at its cut end $\frac{1}{8}$th inch across. It possessed two valves only, an anterior and a posterior, well formed, of equal size, but smaller than the aortic valves (*vide* Plate V, fig. 3).

Following the direction of the pulmonary artery downwards into the right ventricle and below its posterior cusp the muscular wall, an offshoot from the septum, ended abruptly by a rounded edge $\frac{1}{4}$th inch below the lowest attached end of the posterior semilunar valve (*vide* Plate V, fig. 3). In breadth it was perhaps a trifle wider than ¼th inch, and it stretched across the right ventricle from the septum to its anterior wall.

Looked at from an opening in the bottom of the ventricle this muscular barrier divided the cavity into two parts, viz. one part in connection with the tricuspid valve and the aorta, the other part leading to the pulmonary artery, of which the area leading to this vessel was quite a small affair, not much larger than the pulmonary artery itself. This muscular spur was nearly ¼th inch thick.

Viewing this structure on the side of the aorta, in front it formed a V-shaped union (*vide* Plate IV, fig. 1) with the septum ventriculorum to its left, and to its right it ended under the anterior aortic cusp, and on which the whole of this valve was seated. The aorta, therefore, in part arose from this spur. The remaining part of the aorta took origin from the right ventricle mainly, though the bulk of its channel lay in the direction of the left chamber.

The anterior curtain of the mitral valve, which was opaque and somewhat thickened in part, originated in the fibrous attachment in which the tip of the septum ventriculorum ended posteriorly. The anterior cusp of the mitral valve thus shut off the left auricle from the right ventricle.

The anterior tricuspid curtain also in part took origin from this fibrous attachment of the septum ventriculorum. Close to this fibrous attachment on the top of the interventricular septum were some tendinous threads which passed into this cusp.

The posterior segment also in part took origin from the fibrous attachment as well as being fixed by some fibrous threads at the top of the septum.

In the septum between the two auricles was an oblique channel of the capacity of an ordinary school slate pencil. It was guarded on the left by a fibrous valve (semilunar valve) of some thickness, attached above and below to the wall of the left auricle, its free and rounded and rather concave edge projecting into the left auricle. Behind, it

DESCRIPTION OF PLATE IV.

To illustrate Dr. George Carpenter's Two Cases of Congenital Heart Disease.

FIG. 1.—The left ventricle has been laid open and the aorta also. Note stick to the left passing down the pulmonary artery—it has been bent, and its lower end is just visible in the right ventricle. The middle stick has been passed *viá* the right auricle into the left ventricle through the tricuspid valve. Note the cusp of the tricuspid valve arising from the tendinous continuation of the ventricular septum. The fleshy ventricular septum has been cut across and part turned to the right. Note the aorta arising from the right ventricle as well as the left. The stick on the right is passed through the mitral valve from the left auricle. (From photograph by the author. Natural size.)

FIG. 2.—The right ventricle has been opened and displays a cusp of tricuspid valve and an enlarged pulmonary artery dividing right and left (hair-pin passed through) to the lungs. Above this is the ductus arteriosus. A piece of wood can be seen at the bottom of this, which has been passed through the thoracic aorta. The narrow sticks of wood mark the blood-vessels arising from the aorta, which is lost to view behind the pulmonary artery. (From photograph by the author. Actual size.)

PLATE IV.

Fig. 1.

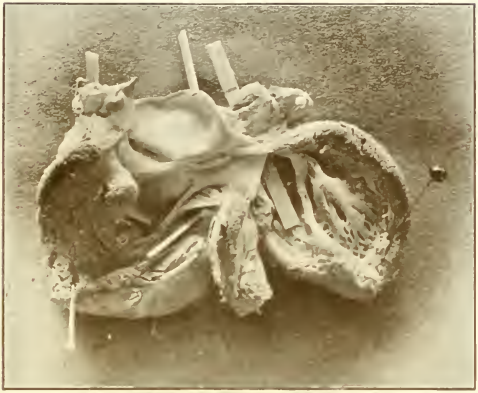
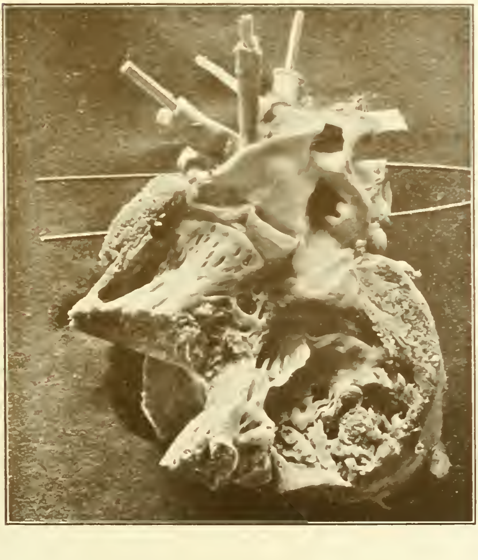


Fig. 2



Dr. George Carpenter's "Two Cases of Congenital Heart Disease."

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DESCRIPTION OF PLATE V.

To illustrate Dr. George Carpenter's Two Cases of Congenital Heart Disease.

Fig. 3.—The left ventricle has been opened and shows the small pulmonary artery with two valves—one cusp distended by a piece of wood. To the left of this is the aorta. A stick has been passed along this into the right ventricle in front of the tendinous cords of the tricuspid valve. This stick will be seen to be obviously bent. The long stick at the top of the figure has been passed *viâ* the right auricle, through the tricuspid valve into the right ventricle. It is lying deeply. The top stick to the right is passing through the left auricle into the left ventricle through the mitral valve. (From photograph by the author. Natural size.)

Fig. 4.—The left ventricle and left auricle have been opened by a lateral vertical incision. Note in the foreground the cusps of the mitral valve, behind this a dark hole, the opening of the tricuspid valve with the cusps surrounding it. Below this is the top of the ventricular septum. Above the black hole is the inter-auricular septum translucent below, opaque above, with fenestræ posteriorly. (From photograph by the author. Actual size.)

PLATE V.

FIG. 3.

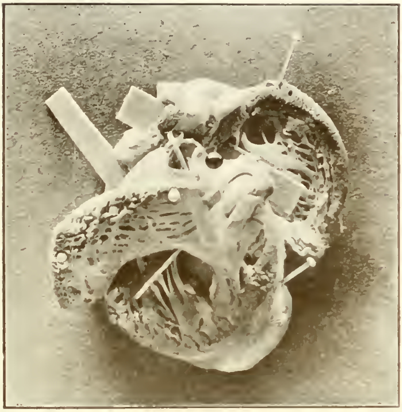
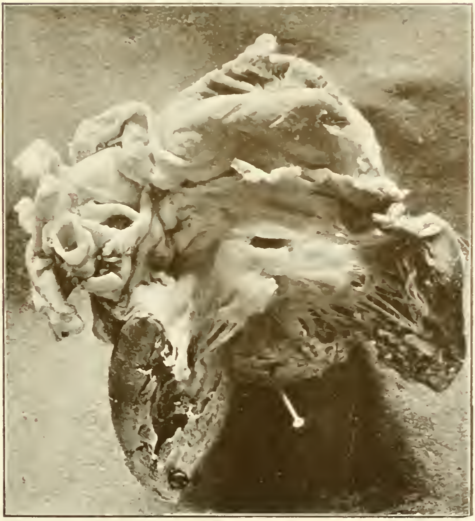


FIG. 4.



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disappeared in the auricular septum, of which it was part and parcel, and with which it eventually blended. On the septum on the right side there was a corresponding depression, the foramen ovale, completely guarded by this semilunar valve. But the valve, although sufficient to cover the foramen, yet displayed a small perforation about the size of a large pin puncture. The two auricles were of about equal capacity and thickness. The heart was inclined to bun shape, and the left ventricle was twice the thickness of the right (*vide* Plates IV and V, figs. 1 and 3).

The pulmonary artery divided into two branches of about equal size, and there was a third patent vessel the size of a silver probe, apparently the ductus arteriosus. A similar patent vessel corresponding to the situation of the ductus arteriosus was found in the aorta, but the arteries were unfortunately severed before the connection had been verified.

The pickled organ weighed three quarters of an ounce.

Liver.—The central parts of the lobules appeared unduly pale, and these microscopically stained poorly in relation to other parts, which took the stain well. The liver-cells were rather ghost-like. There was also over-nucleation in some of the portal canals. The capillaries were plentifully supplied with red blood discs. All other organs were quite healthy.

The tongue did not display any naked-eye abnormality on section.

CASE 2.—*Defective auricular septum; right auricle the much more capacious of the two and twice the thickness; pulmonary artery larger than aorta, the thoracic aorta being practically a continuation of that vessel by the ductus arteriosus; arch of aorta constricted at its junction with the ductus arteriosus.*

Ivy C—, aged 9 months; had wasted for one month. Mother had had nine other children: three miscarriages, three premature, six alive, the others died when a few months old. Twins before the birth of patient, now deceased, and before these a premature birth which only survived a few hours.

On examination.—A small snuffling child, not cyanosed, but, according to its mother, "gets blue." She had snuffled from birth, but there was no discharge by the nostrils.

She had a decided rickety rosary, the wrist ends were enlarged, the anterior fontanelle was widely open, there was no cranio-tabes and the liver and spleen were not enlarged.

Heart.—Apex beat fifth interspace, $\frac{1}{4}$th in. outside the nipple. A systolic bruit was heard loudest at the second left interspace near the sternum, but it was not at all striking (child crying loudly). There was neither thrill nor epigastric impulse. Previous to my examination the house-physician had described "a loud somewhat harsh systolic murmur, best heard over the pulmonary area, but also heard to the right of the sternum and at its upper end." By X-ray examination the right side of the heart was one finger's breadth to the right of the right sternal margin, and the extreme left side was nearly two fingers' breadths to the left side of the left sternal margin. That was on January the 13th, 1908. On the 16th I again examined the heart under more favourable circumstances, and the systolic bruit was loudest over the third left interspace close to the sternum. It was audible all over the chest, back and front, conducted better to the child's left side than to its right, and was heard in the great vessels of the neck. In the interval the child had developed pneumonia, and there was dulness, tubular breathing, and broncophony over the right base. She way very pale and very thin. At 6 p.m. she was admitted into hospital extremely collapsed, and at 9 p.m. her temperature was 107° F. Between this time and 10 o'clock the following morning, when she died, the highest temperature was 106° F. and the lowest 104·2° F. *The child was at no time cyanosed.*

Autopsy.—Pneumonia was found as described during life. The heart was inclined to be rounded on the right side; the left ventricle, although appreciably more muscular than the right, did not bear the usual relative relationship in regard to thickness.

The pulmonary artery was a trifle larger than the aorta. It possessed three semilunar valves of equal size (*vide* Plate IV, fig. 2). It branched right and left into vessels ¼th inch in diameter, and was continued onwards by a vessel which curled over the bifurcated artery and terminated in the aorta.

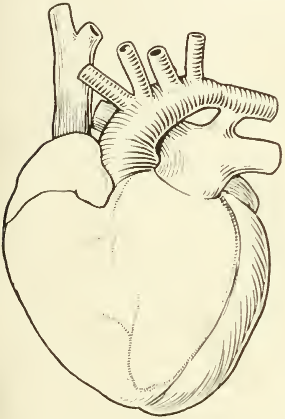


FIG. 42.—Front view of heart, showing continuation of the pulmonary artery (ductus arteriosus) as the thoracic aorta. The innominate artery arising as two vessels. Semi-diagrammatic representation (*vide* Plate IV, fig. 2).

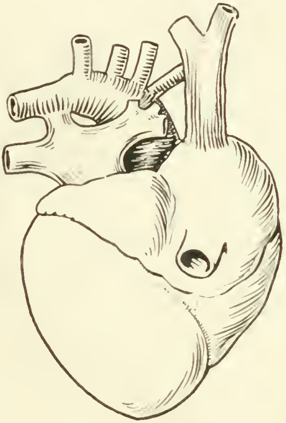


FIG. 43.—Back view of heart, showing continuation of the pulmonary artery (ductus arteriosus) as the thoracic aorta. Semi-diagrammatic representation (*vide* Plate IV, fig. 2).

The aorta was a somewhat smaller vessel than the pulmonary artery. It displayed three semilunar valves of equal size, and above them were seen the orifices of three coronary arteries, two of them being close together. This vessel having given origin to four great vessels instead of three terminated in the thoracic aorta—the continuation of the pulmonary artery (Figs. 42 and 43). At the actual junction of the pulmonary artery and the aorta the latter vessel was narrowed (Figs. 42 and 43), and at this point was somewhat larger than the arteries arising from the aorta. Beyond this union the aorta was smaller across than either branch of the pulmonary artery, being $\frac{3}{16}$ths inch in diameter, and it was a direct continuation of the pulmonary artery rather than of the aorta.

The thoracic aorta was therefore supplied by the aorta and pulmonary artery, while the great vessels of the head and neck and upper extremities were filled by the aorta.

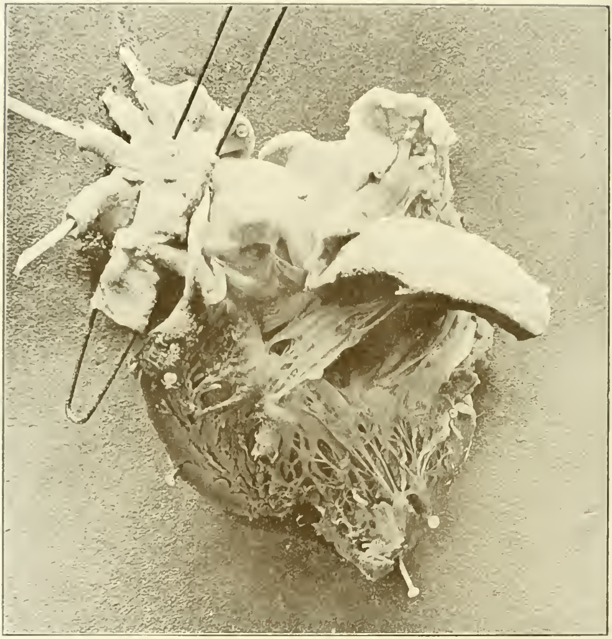
The right and left auricles practically formed one large chamber, the right side being much the more capacious. The auricular appendix on the right side was a large affair and part and parcel of the auricle, whereas that on the left side was small and rudimentary.

The auricular septum was represented by a translucent membranous partition of semilunar outline hanging from the roof attached back and front, and with the arc or bow at the top, and with the base of the bow hanging free in the chamber and falling far short of the ventricular septum (Plate V, fig. 4). It measured slightly more than $\frac{1}{4}$th inch in depth, and about $\frac{5}{8}$ths inch from before back. Posteriorly it was a trifle fenestrated. The right auricle was about twice the thickness of the left. Looking into the chamber on the right side was seen the trefoil arrangement of the tricuspid valves, and on the left the two leaves of the mitral valve. Between them, wall-like, lay the upper rounded edge of the septum ventriculorum, which here ended free, and from the sides of the wall sprang the cusps of the tricuspid and mitral valves.

On looking into the left ventricle the aortic cusp of the mitral valve was seen to extend as far as the origins of the semilunar valves, the posterior and the external.

The top of the ventricular septum was seen to pass from before backwards and obliquely downwards. The top of the fore part was muscular and joined the aorta at

PLATE VI.



Heart of a child aged 9 months. The left side has been opened and a wedge-shaped section of its wall turned up and back, which displays the following features: The ventricular septum is partly muscular and partly membranous, and arising therefrom are the two curtains of the mitral valve. At the top left-hand corner a hair-pin has been passed along the right and left branches of the pulmonary artery (*vide* also Plate IV, fig. 2). The bottom stick of wood marks the commencement of the thoracic aorta. For further particulars see text. (From a photograph by the author slightly enlarged.)

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the external (partly) and the anterior aortic valve where it met the undefended space. Behind this part it was membranous (Plate VI). Here the aorta and the anterior wall of the auricle and the fore-end of the membranous septum were united, and at this spot the muscle of the ventricle terminated. Viewed by transmitted light there was seen a definite ring, the commencement of the aorta. From this ring hung a membranous curtain, the aortic flap of the mitral valve, which was attached on either side. On one side it was fixed by tendinous chords to the top of the ventricular septum, which here rapidly fell away, leaving a large membrane occluded area. On the other side it was attached to the side of the ventricle directly and by papillary muscles. This curtain, although much broken up into tendinous chords, was nowhere perforated, and it appeared to readily shut off the ventricle and the auricle. On the opposite side the tricuspid flap arising from the ventricular septum and the adjoining auriculo-ventricular ring aided in the separation of the two ventricles, and the tricuspid valve was efficient. The pulmonary artery, unlike the aorta, arose entirely from muscle; that of the right ventricle and its chamber was a direct communication with it. Although the aorta was in direct communication with its ventricle, the cavity of the ventricle was smaller and the aortic orifice was narrower.

Remarks.—Neither of these infants was cyanosed, although in both cases there must have been a free admixture of arterial and venous blood.

So often does this happen in cases of congenital malformation of the heart, and so infrequently is it attended by cyanosis, that it hardly seems worth while to seriously consider a theoretical explanation for the blueness, which affords so little corroboration in practice.

Cyanosis is an indication of deficient aëration, not of the admixture of arterial and venous blood.

The addition of extensive consolidation of the right lung in the second case was not sufficient to curtail its complete oxygenating functions. Although congenital heart disease and morbus cæruleus are often viewed as inseparable partners, yet this is by no means the fact, as frequently cyanosis does not appear for months or even years. It often arises after some acute illness, such as measles, scarlet fever, typhoid, bronchitis and diarrhœa.

The most common murmur in congenital malformation of the heart is a systolic bruit, which is often audible all over the chest, back and front, and is best heard over the pulmonary artery or along the left side of the sternum at some point between the pulmonary artery and the junction of the xiphoid and manubrium sterni or at that spot. This is a murmur which occurs with stenosis of the pulmonary artery or with a perforate septum ventriculorum, either separately or in combination. Further, when the septum is perforate the murmur is usually audible in the great vessels of the neck, but not otherwise.

When once present it always persists, but even then varies in intensity from time to time. But while one is perhaps warranted in diagnosing malformation of the pulmonary artery in children beyond the age of two years when such a murmur is detected, yet the cases just selected illustrate the fact that in infants, at any rate, systolic bruits of greatest intensity over the pulmonary artery or between there and the xiphoid cartilage in the vicinity of the left margin of the manubrium sterni do not permit the same deductions being drawn from them. It is during the first few months of life that those with the more pronounced cardiac malformations succumb to their abnormalities, and it is not until these have been eliminated that such sounds, if heard on auscultation, will be likely to be correctly interpreted by the diagnosis of malformation of the pulmonary artery or defects of the ventricular septum.

While in infants the detection of a loud systolic bruit may be looked upon as the most reliable and most constant, and often the only obvious, sign of congenital malformation of the heart, yet, in the present state of knowledge, deductions drawn from the character, the point of greatest intensity, and the conduction of the bruit are most untrustworthy and most misleading.

(*Exhibited February the* 21*st*, 1908.)

Discussion.

The Chairman (Dr. Edmund Cautley) said that when specimens from congenital heart cases were shown before the Society they sometimes contradicted the diagnosis made during life. It would be well if a collective investigation were made as to the true significance of congenital heart murmurs, and all possible cases exhibited, especially those which were likely to die, so that the specimen could be shown later. That would place the diagnosis of such murmurs on a more satisfactory basis. Dr. Carpenter believed the right auricle in one of his cases was practically absent, but his own view was that it was only smaller than usual.

Dr. F. J. Poynton said that cases of patent ductus arteriosus were described as having a long murmur heard to the left of the sternum, which occupied the systole and diastole, as a sort of continuous humming murmur. But it had also been recorded by the late Sir William Broadbent and others that the same continuous murmur was associated with patent septum ventriculorum. He asked whether Dr. Carpenter thought the murmur in his second case was exactly like the murmur in his first case, or whether it struck him that it occupied the whole systole and diastole. He would also like to hear if any member had made an autopsy on a case where there was a continuous humming murmur over the pulmonary region. Cases of the kind which he had seen had done remarkably well, living with few bad symptoms. He had three which were alive without any cyanosis, though with some shortness of breath.

Dr. William Ewart hinted at the possibility that in some of the cases previously described the murmurs in question in reality have been intra-thoracic venous murmurs. But in Dr. Poynton's cases the cardiac character of the murmur seems to have been so pronounced as to have left no room for any doubt in that direction.

Dr. Charles W. Chapman said such cases met with in children did not seem to die, yet the murmur did not appear to be met with at a later age. He did not know what became of them. Some of the patients in whom there was a continuous humming went about quite well and there were no symptoms. If he was able he would bring some of these cases before the Society.

Dr. George Carpenter, in reply, said the murmur in both cases was exactly similar to that which was audible in stenosis of the pulmonary artery; of that he had no doubt. He thought the systolic murmur produced in stenosis of the pulmonary artery without a patent septum ventriculorum and with a patent septum ventriculorum were identical. Certainly a perforate septum ventriculorum produced a systolic murmur the same in character as that observed in pulmonary stenosis. He was once under the impression that if the murmur were conducted into the big vessels of the neck one could diagnose patent septum ventriculorum, but the more he saw of congenital heart malformation in infants, the more difficult he found it to decide by auscultation what lesions were present. He was well aware that a continuous humming sound over the pulmonary area was described as occurring in patent ductus arteriosus, but he had had no clinical experience of such a case. In his first case there were several possible sources of bruit production: (1) A small perforation in the semilunar valve; (2) patent ductus arteriosus; (3) a small pulmonary artery; (4) aorta arising from both ventricles above the ventricular septum. He did not think the murmur was produced at the semilunar valve as it was systolic in point of time, and doubtfully not at the patent ductus arteriosus for the same reason. He intentionally said "doubtfully" because he thought it not unlikely that a systolic bruit might be caused by such a malformation, especially if associated with narrowing. In the second case the possible sources of production of the murmur were: (1) The defective auricular septum; (2) the enlarged pulmonary artery; (3) the patent ductus arteriosus and narrowed junction with the aorta. Neither in this case did he think the systolic bruit was produced by the defective auricular septum. The possible sources were either the small pulmonary artery or the patent ductus arteriosus. The murmur was neither humming nor continuous but definitely systolic.

48. FRONTAL MENINGOCELE, CEPHALHÆMATOMA, AND MYELO-MENINGOCELE IN LUMBO-SACRAL REGION.

By EDMUND CAUTLEY, M.D.

The specimens were from a male infant who had died at the age of eighteen days. He was the seventh child, and the mother had had no miscarriages. There was nothing in the history to suggest inherited syphilis. Labour was difficult and instrumental.

The child was admitted into the Belgrave Hospital for Children on the seventh day of life for swellings which had been noticed since birth. In the mid-frontal region, rather more to the left than to the right of the middle line, was a soft projective swelling about the size of half a walnut. It could be reduced, and became more tense on crying. It was undoubtedly a meningocele protruding through a roughly circular orifice an inch in diameter. The skull-cap showed that this orifice was completely closed by the dura mater, and it was noticeable at the autopsy that the local swelling had disappeared. In the left posterior parietal region over the upper two thirds of the bone there was another more diffuse swelling, which was not reducible and only became slightly more tense on crying. Pressure at the posterior border enabled one to feel an irregular raised edge as if a portion of the bone was depressed. The skull-cap showed that this irregular edge was possibly an abnormal suture, and that the swelling was a hæmatoma. In addition to the effusion of blood under the periosteum there was a corresponding, but smaller, effusion between the dura mater and the bone; and a like subdural effusion on the opposite side. Patches of cranio-tabes were also visible in the posterior halves of the parietal bones. These patches suggested imperfect or delayed ossification rather than softening. In the lumbo-sacral region there was a swelling about the size of half a Tangerine orange. It was covered with skin and a thin corrugated membrane at its summit and had discharged a scanty serous fluid since shortly after birth. The arches of five of the vertebræ were deficient. The cord was much attenuated and could be traced from the spinal canal into the swelling to its lowest extremity, from which nerves were given off. The tumour was undoubtedly a myelo-meningocele. The child presented no other peculiarities except bilaterial talipes. He died from asthenia. (*Exhibited February the* 21*st,* 1908.)

49 CEREBRAL ATAXIA AND IMBECILITY.

By EDMUND CAUTLEY, M.D.

This patient is interesting from the point of view of diagnosis and prognosis. He is under observation in the Belgrave Hospital for Children.

He has been deserted by the mother. The child's present guardian states that the mother has had two miscarriages, two stillborn children, and another child which is now three years old.

Although he is two years and one month old he only weighs 14 lbs. 14 oz., has a small head, measuring 17¼th inches in its largest circumference, and is unable to stand or walk, cannot talk, and only uses his hands slightly. He feeds himself with food such as biscuit and bread, but will not use a spoon. When he uses his hands there is a moderate coarse tremor of the hands and arms. He can maintain the sitting posture, but does not pull himself up into it. The reflexes are exaggerated, especially the knee-jerks. There is no rigidity. Mentally he does not look imbecile, yet there is no doubt that he is lacking in intelligence.

The case belongs to the group of birth palsies, the result of delayed development, injuries at birth, meningeal hæmorrhage, or encephalitis. In this particular instance the main damage has been in the frontal region and consequently the intellectual faculties are impaired. The ataxia is probably due to slight involvement of the arm centres.

The prognosis is doubtful. Probably the child will always be backward or feeble-minded, but as he gets older it may be possible to educate his motor and psychical functions. (*Exhibited February the* 21*st*, 1908.)

Discussion.

Dr. Porter Parkinson thought there was much room for differences of opinion about the case. He suggested the possibility of syphilitic meningitis. With regard to rickets, the fontanelles were not closed, but the head was small, and in rickets he found the head to be a little larger than usual for the age. Syphilitic meningitis most usually affected the base, and often caused hydrocephalus, but he did not think that had happened here. Syphilis might cause more than one lesion in a case, and there may have been some encephalitis as well as meningitis, not acute, but taking the form of a fibrosis and leading to arrest of development of the brain-tissue

The Chairman (Dr. Edmund Cautley), in reply, said he thought the case belonged to the group of cerebral diplegias; that it was due to injury at birth causing either meningeal hæmorrhage or encephalitis; or, less probably, to delayed development—the so-called "agenesis corticalis." In that instance the main incidence of the disease was on the prefrontal convolutions, the motor area being very little affected. But that it was affected was shown by the ataxia in the hands and arms, and perhaps also by the delay in learning to walk. He thought the child would always be more or less backward, though the amount of imbecility was difficult to estimate at present. She would be able to walk, for there was no muscular rigidity, and as she was now somewhat intelligent her mental capacity might become fairly good.

50. ANGEIOMA OF RIGHT AURICLE AND MEATUS.

By MACLEOD YEARSLEY.

This patient was first shown by me at a meeting of the Otological Society on March the 6th, 1905. She was then 10 years of age.

The growth has greatly increased in size since that time, but the parent persistently refuses an operation. I show you the case to-day as an interesting example of an angeioma. Three years ago the right auricle was only slightly larger than the left; now it measures 3 by 2 inches whilst the left is only 2⅜ths by 1½ inches. The growth extends down the meatus nearly to the membrana tympani. Dr. Seligmann, the pathologist to the Zoological Society, informs me that he has observed similar growths in the auricles of fox-terrier bitches. I propose to dissect it out; it is not a case for electrolysis.

(*Exhibited February the* 21*st*, 1908.)

51. SPECIMEN FROM FATAL CASE OF LABYRINTHINE SUPPURATION.

By MACLEOD YEARSLEY.

E. R—, aged 13 years, a scholar at the L.C.C. Residential Deaf Centre for Girls, was first seen by me at the Royal Ear Hospital on December the 4th, 1907. She had become deaf at the age of 3½ years from double suppurative otitis media following scarlet fever. She was blind in the left eye from corneal ulceration, which had resulted in an anterior synechia.

Her condition when I first saw her was as follows: Right ear, very large perforation with a small polypus. Left ear, membrane almost wholly destroyed, pus coming from the attic. Her hearing for the voice was 0 both ears. She had, however, retained very good speech.

Antiseptic treatment was ordered until she could be taken into hospital.

On January the 23rd a radical mastoid operation was performed on the left ear. The attic and antrum contained much granulation tissue, the carious head of the malleus was found, and there was caries of the long process of the incus.

The polypus on the right side was removed.

The patient did well until January the 26th, when the evening temperature rose to 100·6° F., coming down to normal after the first dressing on the 27th. On the 30th the temperature again rose to 102° F., and on that afternoon the radical mastoid operation was performed on the right ear. No ossicles were found. The roof of the tympanum and antrum was found to be carious and was removed, exposing an intact dura mater. The inner tympanic wall was explored and the oval window appeared to be closed by cicatricial tissue. The ear was packed and the wound closed in the usual way.

On January the 31st the temperature was 103·8° F. in the afternoon, the patient was drowsy, vomited a good deal, and had right lateral nystagmus. The patellar reflexes were absent, but there was no rigidity. There was a tendency to lie curled up. She was given a powder containing gr. x of antipyrin with gr. v of calomel and the dressings changed. The temperature fell to 99° F., but rose again at 10 o'clock on February the 1st. The wound was then re-opened and the dura found to be bulging and non-pulsatile. More bone was removed and the dura opened. Straw-coloured fluid escaped and pulsation was re-established. The temporo-sphenoidal lobe was explored without result and the operation cavity was packed from the wound.

The temperature fell to normal and at 4.30 the patient was much more comfortable. There was still right lateral nystagmus. At 6 p.m. she was worse again, the temperature being 106·6° F., pulse-rate 96 to the minute, and respirations 42. At 8 o'clock she had a definite rigor, the temperature reaching 104·2° F. She was very noisy all night, in spite of morphine, and at 1 a.m. began to pass everthing under her. On February the 2nd the brain was again explored and a flow of very foul coffee-ground-like fluid, about 1½ to 2 oz., was evacuated from a direction forwards and inwards. This was taken to be a cerebral abscess at the time and a drain was put in.

No improvement resulted, she continued semi-delirious, the temperature reaching normal at 11.30 p.m. She complained at intervals of severe head pain, and there was some amount of retraction of the head. The temperature gradually rose on December the 3rd, until it was 103·4° F. at 6 p.m., when she died. At no period was any optic neuritis detected.

A post-mortem examination was made on February the 4th by the pathologist, Mr. Bowen, the head only being examined in deference to the wishes of the friends.

There was diffuse suppurative meningitis, the site of infection being a small erosion with a corresponding perforation in the dura mater in the neighbourhood of the superior semi-circular canal.

(*Exhibited February the 21st, 1908.*)

Discussion.

Mr. H. S. Clogg asked to what class of case the treatment mentioned was applicable.

Mr. Macleod Yearsley, in reply, said he had not yet made up his mind what class of case the treatment would apply to. If there were disease in the semi-circular canals he thought that by opening up the vestibule in the manner described much could be found out by means of a fine probe, and he was inclined to do this in such cases, and he could work his way into the cochlea if necessary. He had tried it on skulls, and it would certainly drain any disease above the opening in the vestibule.

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52. A CASE OF BRONCHIECTASIS.

By T. R. WHIPHAM, M.D.

The patient is a boy, aged 16 years, but looking much younger. He had a severe attack of bronchitis and pneumonia when 14 months old, and apparently has been affected with his chest since, though his symptoms are said to have caused him no distress until last Christmas. Since then he has suffered from a good deal of cough and expectoration, the latter being especially copious in the mornings, and has been very short of breath. The boy presents a marked cyanosis; the face is congested and the lips and ears are blue. The finger-nails are curved and cyanosed, and the finger-tips are somewhat clubbed. The chest is very deep antero-posteriorly, and the back is bowed, his attitude being like that of an old man. The movement of the chest is very limited, there being scarcely any lateral expansion, so that respiration is carried on almost entirely by the upper ribs and the diaphragm. His breath at times is excessively offensive. The whole of the left side of the chest, both in front and behind, yields a poor percussion note, and the upper part of the right lung posteriorly is also somewhat dull. The breathing over these areas is slightly diminished in intensity, but patches of amphoric breathing, which vary from time to time, can be heard at the left base behind and over the left upper lobe in front: a small patch of similar breathing has also been heard at the angle of the right scapula. Creaks and râles are audible over both sides, especially on the left. The heart is displaced to the left, the apex being beyond the nipple line, while the right border of the dulness does not extend beyond the left edge of the sternum. To the X rays the left side of the chest is abnormally opaque throughout, especially at the base, and on the right side there are patches of dulness, as of fibrosis of the lung and of an enlarged mediastinal gland. His red blood-corpuscles number 3,420,000 per c.mm.; there is thus no polycythæmia, as is often found in such cases. His sputum is copious, thick and purulent, and has been found to contain tubercle bacilli.

(*Exhibited February the* 21*st*, 1908.)

Discussion.

The Chairman (Dr. Edmund Cautley) asked what Dr. Whipham considered was the prognosis in the case, and his advice as to treatment. Bronchiectasis, even apart from tuberculosis, was, in his experience, very difficult to treat satisfactorily, and some cases went from bad to worse, no matter what was done for them, whereas others seemed to get well whatever treatment was adopted.

Dr. T. R. Whipham said his prognosis was a bad one. The boy, who was employed in a warehouse, had been fairly well until last Christmas, but since then he had become rapidly worse. He thought that bronchiectasis was the primary lung condition, and that tubercle had been superadded. He had only seen him since Christmas, and he could not say whether his sputum contained tubercle bacilli or not before that date. Bronchiectasis in children he always looked upon with grave suspicion. He had nothing to add to what was already generally known on the treatment of such conditions. In answer to a suggestion that it might not be bronchiectasis, he said that such a condition need not always be bilateral. He showed before the Society last year a child with marked signs of bronchiectasis on one side of the chest, and, as he thought, slight signs on the other side also. That child died in the Belgrave Hospital, and there were found, post-mortem, multiple bronchiectases in the one lung, while the other was merely emphysematous.

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53. A CASE OF INCOMPLETE CONGENITAL OPHTHALMOPLEGIA EXTERNA, WITH REMARKS.

By SYDNEY STEPHENSON, C.M.

Instances of congenital defects in the ocular movements, although certainly not common, must be sufficiently familiar to every ophthalmic surgeon and neurologist. Any and every extrinsic muscle may be involved, and combinations of all sorts may occur. One or both eyes may be affected, usually the latter. Congenital ptosis, which is, of course, an example of the condition, is the defect most frequently met with, and when present, produces so striking an alteration in the patient's appearance that it cannot be overlooked, even by the most inattentive observer. Defects in the mobility of the eyeball are often associated with congenital ptosis, as in the case to be immediately described. Next to the levator palpebræ superioris the commonest muscle to be affected is the external rectus, usually on both sides. Paralysis of other cranial nerves, particularly of the seventh, may be associated with the defective mobility of the eye, as in cases reported by Heubner, Warrington, and others.

Congenital ptosis must be carefully differentiated from the ptosis that may occasionally be the direct consequence of injury at birth by forceps or otherwise. Thus, Berger ('Arch. f. Augenheilkunde,' 1887) after a difficult forceps delivery (instrument slipped twice) observed paralysis of the levator palpebræ superioris and the superior rectus. Again, in Bruno Wolff's elaborate monograph on 'Injuries to the Eyes of the Child during Labour' (translated in the 'Ophthalmoscope,' September, 1907, to February, 1908) no fewer than seventeen cases are quoted where paralysis of the extrinsic muscles of the eye were apparently due to injuries received during birth.

The facts of my own case are as follows:

Israel H—, a Jewish boy, aged 8 years, was brought to the Evelina Hospital on February the 11th last by his father, who stated that the boy had never been able "to open his eyes," and that the condition tended to become worse towards nightfall. There was no family history of any similar defect.

Personal history.—The child, born at term, was delivered by instruments, the scalp being wounded during the operation. When six weeks old he developed a rash in the so-called "diaper region." For the first two years of life the child ailed on and off, suffering from "wasting," diarrhœa, and so forth. There were never any "fits."

Family history.—There are two younger children, both believed to be strong and healthy, and neither known to have any abnormality of eyes or of sight. The father admits having suffered from "chickenpox" on the penis when eighteen years of age, that is to say, before ever he had sexual connection. No account of secondary symptoms can be obtained. The mother, according to the husband, is a delicate woman, who complains of her sight, but who has never worn glasses.

On admission.—A fairly well-nourished, but rather dull-looking boy, with "adenoid" facies. His mental development, however, is average. No syphilitic stigmata can be found in face or teeth. In a medium light the transverse diameter of the pupils measures 6 mm. They respond to light directly and indirectly, although the reaction in the right is less than that in the left eye. Response to accommodation equal and normal. No corneal opacities; no Hirschberg vessels. There is incomplete but bilateral ptosis. The movements of both eyes are deficient in all directions. The greatest range is downwards, while the excursions outward, inward, and upward are less than 2 mm. in range. R. V. $\frac{6}{24}$ and No. 1 Jaeger. L. V. $\frac{6}{60}$ and No. 1 Jaeger. Refraction, estimated while the eyes were under the influence of atropine, was R. E. $\frac{+\ 0{\cdot}25}{+\ 2{\cdot}75\ 90^\circ} = \frac{6}{12}$; L. E. $\frac{+\ 1{\cdot}0}{+\ 3{\cdot}0\ 90^\circ} = \frac{6}{24}$. There is a slight and incomplete form of coloboma of the optic discs, more marked in the left than in the right eye.

No evidence of paralysis of the seventh cranial nerve. Knee-jerks absent, even on re-inforcement. On the other hand, the ankle-jerks appear to be present. No scars can be found upon the scalp or face in token of instrumental delivery.*

Remarks.—In the foregoing case, then, there is defective movement as regards the extrinsic muscles of both eyes supplied by the third, fourth, and sixth cranial nerves.

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* For these notes I am indebted to Dr. J. Sydney Pearson, my clinical assistant in the Ophthalmic Department of the Evelina Hospital.

According to the anamnesis, the condition has existed since birth. It is of some importance to observe that in this case the extrinsic muscles of the eyeball are alone involved, while the intrinsic muscles are intact. In point of fact, it is uncommon for the latter to participate in the defect, although that is very different from asserting, as a recent writer on nervous disease has done, that "No evidence of an affection of the internal ocular muscles has ever been present" ('Paralysis and Other Diseases of the Nervous System in Childhood and Early Life,' by James Taylor, 1905, p. 349). Indeed a few years ago I brought under the notice of this Society ('Reports,' vol. ii, 1902, p. 117) a case that proved the contrary. The patient, a girl, aged 6 years, presented almost complete paralysis of the right oculo-motor nerve, both internal and external branches being affected. The dilated pupil, which measured 5 mm. transversely, had no action to light. It became smaller after the local application of physostigmine (eserine), and larger after homatropin. It was, in other words, an instance of congenital and total ophthalmoplegia. Moreover, a case of congenital ophthalmoplegia interna has recently been published by Dr. Georg Levinsohn, of Berlin ('Klinische Monatsblätter für Augenheilkunde,' xlv, 1907, p. 391). The patient, a man, aged 17 years, presented as a congenital defect pupils dilated almost *ad maximum*, which were motionless both to light and to convergence, so that the eyes looked much as if atropine had been dropped into them. The punctum proximum lay at 30 cm. from the right, and at 25 cm. from the left eye. The pupils responded to the use of physostigmine. The movements of the eyes were good. So far as I know, Levinsohn's case of almost complete bilateral ophthalmoplegia interna is unique. We may conclude, then, that although ophthalmoplegia interna is rare as a congenital defect, yet it may occur not only associated with a defect in the extrinsic muscles of the eye but also as an isolated symptom.

The influence of heredity in the production of congenital ophthalmoplegia is often most marked. In Gourfein's remarkable case ('Soc. d'anatomie et de Phy. de Bordeaux,' May the 18th, 1903) six members of the same family (paternal grandmother, father, and four of six children) were affected. In a case published by Mr. J. B. Lawford ('Trans. Ophthal. Society U. K.,' vol. viii, 1888, p. 262) a father and four of seven children suffered from a congenital defect of the ocular movements. Less striking examples of heredity have been published by Hirschberg, Homen, and others.

As regards the explanation of cases such as my own there is no unanimity of opinion even among those best qualified to judge. The two chief theories of causation are—(1) muscular, and (2) nervous.

(1) The advocates of the muscular theory claim that defects in movement are due to the absence, incomplete development, or defective insertion of the particular muscle or muscles involved. There is anatomical evidence in support of this view. The leading case (as the lawyers say) is perhaps that of Heuck ('Klinische Monatsblätter für Augenheilkunde,' 1879), who made an autopsy on the body of a man aged 18 years. During life this individual had exhibited ptosis with absence of the upward and downward movement of the eyeballs, together with defective action of the internal and external recti. The levator palebræ superioris was badly developed on the right side and absent from the left, thus accounting for the ptosis observed during life. The imperfect movements of elevation and depression were apparently due to an abnormal insertion of the superior and inferior recti muscles. Indeed, in Heuck's curious case every extrinsic muscle of the eyeball, with the single exception of the internal rectus, was inserted into an abnormal part of the sclerotic coat of the eyeball. The muscular theory, in brief, is doubtless capable of explaining satisfactorily many of the cases where a single ocular muscle is congenitally paralysed.

(2) The nervous theory of causation assumes that the

defective movements of the eyeball are due to absence or functional inactivity of the nuclei of origin of the nerves which supply the affected muscles. This nuclear palsy, for that is what it amounts to, may presumably be due either to mal-development or to intra-uterine inflammation. In my patient it is tempting to speculate that the absent knee-kerk implies a latent syphilitic taint, which was responsible for a polio-encephalitis during fœtal life. This hypothesis (I employ the word advisedly) would at all events explain the state of the ocular muscles. The evidence in favour of a nuclear origin for congenital ophthalmoplegia is partly observational and partly inferential. To take the former first, Wilbrand and Sänger ('Die Neurologie des Auges,' 1900, vol. i, p. 87) found more or less complete aplasia, that is, imperfect development, in the third nerve nuclei on both sides in a man who was born with ptosis, and who died at 47 years of age from phthisis pulmonalis. Again, Heubner ('Berlin Medical Society,' May, 1900) obtained a post-mortem examination on the body of a child, who, during life had been affected with paralysis of both sixth and seventh nerves, together with incomplete paralysis of the left third nerve and absence of the lacrimal secretion. The brain of this infant showed no changes, macroscopic or microscopic. The medulla was incompletely developed, and manifested an atrophy extending from the olivary body to the nuclei, the lesions being more pronounced on the left than the right side. The inferential evidence in favour of the nervous origin of congenital defect of ocular movement is, to my mind, of a strong order. To begin with, there is the important observation made by several writers, as Möbius, Fryer, Lawford, and Schapringer, namely, that although the internal recti have acted imperfectly, or not at all in association with the external recti, yet little fault could be found with their action during convergence. It is difficult to understand how this could be the case were the defective movement in reality due to absence or mal-development of the muscle in question. Secondly, in a case such as that reported in the present communication it is not easy to believe that a paralysis affecting almost all the branches of three cranial nerves, the third, fourth, and the sixth, could be the outcome of malformation or absence of the six extrinsic muscles of the eyeball supplied by those three nerves. Thirdly, in some instances, at least, examination has shown that the muscles are normal. For example, elsewhere (*loc. cit.*, p. 120) I have reported the case of a girl, aged 16½ years, affected from birth with imperfect abduction of the eyeballs, especially as regards the right eye. There was a convergent strabismus of the right eye, which measured 40 degrees on the perimeter. The external rectus of the squinting eye was exposed, and found to have, so far as could be judged, a normal appearance and a natural attachment to the sclera. Similar were the findings in a second case of "congenital paralysis of the external recti muscles" in a girl, aged 12 years. The fact that in Levinsohn's case of congenital ophthalmoplegia physostigmine caused contraction of the pupil appears to show that the sphincter pupillæ was present, and its nerve-endings were capable of stimulation by the drug. Lastly, there is the suggestive analogy with congenital palsy of other cranial nerves, as the facial, and, as said before, the two conditions may be associated in one and the same subject. The concurrence of imbecility with these congenital defects of movement, noted by Gowers, Heubner, and Sachs, suggests a defect of development.

On the evidence, I am inclined to attribute most cases of congenital ophthalmoplegia to an affection of the nuclear centres which govern the affected movements. This brings the cases into line with the so-called "congenital nuclear paralyses," such as those which are known to affect the facial, the fifth, and other cranial nerves. Whether, as Sachs has suggested ('A Treatise on the Nervous Diseases of Children,' second edition, 1905, p. 516) there exists a close clinical and anatomical relationship between such cases and those of hereditary progressive muscular atrophy I will not take upon myself to say. *(Exhibited February the 21st, 1908.)*

Discussion.

The Chairman (Dr. Edmund Cautley) asked why it was that such a condition occurred, and why it could occur subsequently to birth apart from ophthalmoplegia externa. He recently saw a girl, aged 18 years, with a history of mental disturbance. She had become dull and confused, but on physical examination he could find nothing abnormal except absent knee-jerks and complete paralysis of the internal muscles of the eyes, the pupils being very widely dilated and not reacting to light nor to accommodation. There was no history of diphtheria. The condition improved, and the next time he saw her the pupils were smaller and reacted. He asked whether Mr. Stephenson had seen cases occurring after birth affecting either the external muscles only, or only the internal muscles, and if so, what explanation was offered?

Mr. Sydney Stephenson said, in reply, that he could not answer Dr. Cautley's question except by saying that after diphtheria the internal musculature of the eye might suffer, or sometimes, as in cases published by Dr Parkinson and himself, the external musculature. He really brought the case to invite an explanation from others.

54. A CASE OF PERSISTENT CAPSULO-PUPILLARY MEMBRANE AND HYALOID ARTERY, WITH ATYPICAL DEVELOPMENT OF THE VITREOUS.

By SYDNEY STEPHENSON, C.M.

Florence B—, aged 15 years; first seen at the Kensington General Hospital on February the 5th last.

Family history.—Father died at fifty years from malignant disease, lasting four months. Mother, aged 54 years, in good health. Seven children born alive, of whom six, including the patient Florence, survive. All babies born at term. No history of deformities in the family can be obtained.

Personal history.—When aged 16 months Florence had a "fit," and this was followed by a squint of her right eye, for which she was taken to the Moorfields Hospital. She has worn glasses for eight years. Beyond the condition of the eye there is no deformity.

Present state.—A well-grown and intelligent girl of fifteen years. The right eye is directed inwards and slightly upwards, so that the corneal reflex lies on the outer margin and somewhat below the horizontal meridian of the (undilated) pupil. The right eye is a little smaller than its fellow, as shown by the fact that the transverse diameter of its cornea measures 10·5 mm. as compared with 11 mm. in the other eye. The movements of both eyeballs are good. Vision = hand-reflex, with or without the glasses she wears. Left vision $\frac{5}{12}$, not improved by her glasses.

With the right pupil undilated, several slack festoons may be seen to extend from the lesser circle of the iris, on the one hand, to a plaque on the anterior capsule of the crystalline lens, on the other. When the pupil is dilated, as with atropine, these threads become taut, and form a beautiful example of capsulo-pupillary membrane. Thus, occupying roughly the lower two thirds of the dilated pupil, are many fine interlacing and anastomosing fibres, which originate from the anterior surface of the iris, and are inserted into a C-shaped plaque, more or less pigmented, lying on the anterior capsule of the crystalline lens. Two other congenital peculiarities are also present in the right eye, namely: (1) A persistent hyaloid artery, and (2) atypical mesoblastic development. (1) The hyaloid artery, which does not contain blood, is visible as a dark-coloured cord running roughly in the axis of the eye, its various parts being seen as stronger and stronger lenses are placed behind the ophthalmoscopic mirror. (2) The retinal vessels are sheathed in opaque, white, glistening bands, which in some instances, as shown in the picture, extend for a considerable distance into the fundus oculi and follow the bifurcations of the vessels. The material varies in amount. The vessels themselves, which carry blood, are more or less concealed by the exudation. The latter can be seen with a + 4 or + 6 D. lens. Refraction, hyperopic.

As regards the left eye, a tenuous thread of pupillary membrane, with anchor-ends, crosses the dilated pupil from above downwards at the junction of its middle and outer thirds. Refraction (estimated under atropine) + 2·0 D. sph. with + 2·5 D. cyl. axis 160° $= \frac{5}{5}$.

(*Exhibited February the 21st,* 1908.)

55. A CASE OF INHERITED SYPHILIS, WITH INFANTILISM, BONE, JOINT, AND VISCERAL LESIONS.

By J. PORTER PARKINSON, M.D.

Esther B—, aged 21 years, has a family history suggesting syphilis, seven out of a family of fifteen having died in infancy, but more than this and her personal history are difficult to obtain. When eight years old she had an affection of the eyes, probably interstitial keratitis, of which some corneal scars remain. For some months she has had pain and swelling of the knees and elbows and pains in the bones. The catamenia have never appeared. She is thin and of a *café au lait* complexion and poorly developed, looking like a girl of fourteen. The breasts are undeveloped, the uterus is about ¾ in. long, and there is no hair on the pubes. There has been some slight general œdema. There are some scars on the trunk, and the upper central incisors are pegged. On the forehead is a non-pigmented scar, and the bone beneath is depressed and irregular but not adherent. The thoracic and abdominal organs appear normal. Urine 2 to 3 pints a day, sp. gr. 1010 to 1015, acid, containing from three fifths to a cloud of albumin and many fatty and granular casts.

There is marked thickening of the tibiæ and ulnæ, general, but with some nodular elevations, and these bones are distinctly tender on manipulation. There seems to be little or no change in the other bones. The elbow and knee-joints are distended with fluid and the synovial membrane appears to be somewhat thickened, and some movable hard bodies can be felt in the right knee-joint; the movement of the right elbow is much restricted by shortening of the tendon of the biceps and supinator longus.

The fundi of the eyes are normal.

The fluid in the knee-joints has much diminished, especially in the left one, which has been treated by simple strapping, while the right one, which has been strapped over Scott's dressing, has not diminished to the same extent. I thought the Society would be interested to see this patient, for though she is not a child she illustrates some of the later effects of inherited syphilis, and the degree of thickening of the ulnæ as felt by the hand and shown in the skiagrams is quite unusual.

(*Exhibited February the* 21*st*, 1908.)

Discussion.

Dr. F. J. Poynton said he had attempted, in one of those cases, to obtain the spirochæte, but without success. He drew the fluid off both joints, strapped them up, and she did well and made an excellent recovery. At University College Hospital recently he had a case of Charcot joint in an adult, in which several investigators were able to demonstrate micrococci in the fluid. Nearly all such joints had been found to be sterile. He mentioned these points as having bearing upon the possible nature of arthritis in syphilitic subjects.

Dr. George Carpenter asked whether Dr. Parkinson could say what was the condition of the ovaries. Infantilism occurred with disease of the testicles and with testicles which were immature, and he thought it was possible that disease of the ovaries in this case might be responsible for the infantilism. He thought a rectal bimanual examination might yield some information on that point.

Mr. Sydney Stephenson asked what was the condition of the eyes in the case. Dr. Parkinson had mentioned that there were remains of interstitial keratitis, but not whether the fundus oculi had been examined and if so, what was found. Interstitial keratitis probably never wholly disappeared, and one could always find in people who had suffered from interstitial keratitis at any time of their life very fine vessels in the depths of the cornea—the so-called Hirschberg vessels, from Hirschberg, the Berlin ophthalmic surgeon, who insisted upon their diagnostic importance. It was desirable to examine the cornea in such cases closely, with the aid of a dilated pupil and strong magnifying glass. Superficial corneal vessels were arranged differently, and were found in a variety of other diseases.

Dr. F. Parkes Weber asked whether the author had met with cases in which the tibiæ were as much enlarged as, or even more thickened than, in the present case, but in which most of the other signs of congenital syphilis were absent. He thought it was partly owing to the occasional extreme thickening and bowing of the tibia or femur in cases of congenital (or even acquired) syphilis * that some authorities, especially in France, were of opinion that the "osteitis deformans" of Paget might be a syphilitic disease. He did not think so himself.

Dr. Porter Parkinson, in reply, said he would have the fluid examined for a micro-organism. He had obtained a very good view of the fundus of the eye, and to him there seemed nothing very obviously wrong with it. He looked for changes in the disc and fundus—either retinitis pigmentosa or choroid trouble. He had not seen a case before in which the bone condition was so marked in congenital syphilis as in this case. He had never seen any great amount of periostitis without some other evidence of syphilis. In the boy shown at the last meeting the periostitis occurred first, and he had treated it three or four weeks before synovitis appeared. He had hoped for an opinion as to the treatment of such cases with mercury, *i.e.* as to whether they showed nephritis. The amount of albumin in the urine had somewhat decreased while he was using the mercury, and the œdema had disappeared.

Dr. F. Parkes Weber said Dr. Parkinson had to some extent answered his own question by his statement that the albuminuria diminished under treatment. In cases of syphilitic disease of the kidneys the question of the advisability of giving mercury had to be decided on by the improvement (in regard to albuminuria and other symptoms) which followed a trial of the (mercurial) treatment in question.

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* Dr. Parkes Weber had once been shown a boy, aged 14 years, with extraordinary enlargement of both legs below the knees resulting from a more or less painless chronic hypertrophic osteitis due to congenital syphilis. Compare H. Lorenz's case of "Osteitis Deformans Heredoluetica" (abstract in 'Münch. Med. Wochens.,' 1904, No. 13, p. 589) in a girl, aged 11 years, and F. Spieler's case of "Chronic Osteo-Periostitis with Increase in the Length of the Right Tibia due to Congenital Syphilis in a twelve-year-old child" (abstract in the 'Münch. Med. Wochens.,' January the 16th, 1906).

Dr. George Carpenter said that Messenger Bradley, of Manchester, was the first to treat syphilitic nephritis by mercury with success. Bradley had treated an infant with catarrhal nephritis and obvious congenital lues by mercurial inunctions, and it completely recovered. He had given mercury in syphilitic nephritis in infants without curing them, although he did not make them any worse by it.

56. ENLARGEMENT OF THE UPPER JAW.

By PHILIP TURNER, M.S.

John C—, aged 9 years and 10 months, was admitted into the Belgrave Hospital for an enlargement of the left upper jaw, which had first been noticed about three months before. The swelling has increased in size lately, but has been painless throughout.

The swelling, which occupies the position of the body of the superior maxilla, extends upwards to the margin of the orbit, bulging forwards so as to obliterate the canine and incisive fossæ. The alveolar process is larger than on the right side and the palate is slightly depressed. There is no exophthalmos and no obstruction to the passage of air through the left nasal fossa. "Egg-shell crackling" can be obtained on pressing the anterior surface of the bone. Six teeth are present on each side of the mid-line in the upper jaw. On the right side the first permanent molar is present with the two permanent incisors, the two temporary molars, and the temporary canine. On the left side the teeth present are: two temporary incisors, the temporary canine, two temporary molars, and the first permanent molar. The second dentition is thus considerably delayed, for at this patient's age in addition to the first permanent molar both permanent incisors and the first bicuspid should be present. A skiagraph was taken, the film being placed in the patient's mouth. This shows the two retained permanent incisors of the left side; the lateral incisor has a well-formed root, but that of the central is short and broad, while from it a definite shadow is continued in an upward direction.

The existence of the swelling with "egg-shell crackling" suggests the presence of either a growth, probably a sarcoma, or of a follicular odontome, frequently known as a "dentigerous cyst." The short history suggests the former, but the non-eruption of the incisor teeth, together with the appearance of the central incisor in the skiagraph, point to the latter as the correct diagnosis.

(*Exhibited February the 21st,* 1908.)

Discussion.

Mr. Russell Howard thought another possibility was myxomatous tumour of the upper jaw. He had a case in hospital two weeks ago in which exactly similar physical signs were present. He took it for a dentigerous cyst, but it was found to be a myxomatous tumour. It had spread from the upper jaw, eroded the orbital plate and the superior maxilla, and had come forward on to the cheek. He scraped the whole of the tumour away and packed the wound, and at present the case was doing well. He did not agree that dentigerous cysts were very rare.

Mr. H. S. Clogg said that in addition to the outer wall of the alveolus being protruded there was a bulging of the inner wall also, which made the alveolar process in that region thicker than it should be. That seemed to him to favour it being a neoplasm of a sarcomatous nature, especially as it had grown somewhat rapidly.

Mr. Lockhart Mummery suggested that transillumination should be tried before going further, as that would show whether the antrum was clear. The thickening might be due to a number of teeth on that side having been arrested, and that for some reason the temporary teeth had not come out. He saw one child with practically two complete sets of teeth due to that cause. He did not think there was a cyst present.

57. MULTIPLE ARTHRITIS.

By PHILIP TURNER, M.S.

The patient, a girl, aged 10 years, had six months ago a painless swelling of the right knee-joint. This was soon followed by similar trouble in the left knee-joint ; shortly after both wrist-joints and the first inter-phalangeal joints of both hands were similarly affected. There has never been any constitutional disturbance and there has never been any pain in the affected joints, though the fingers have been stiff and the other joints have felt weak.

Both knee-joints are now markedly distended and the ends of the bones appear to be enlarged. On each side of the patella thickened synovial fringes can be rolled under the fingers, and the contents of the joint are soft and elastic. Both wrists are enlarged, and there appears to be fluid in the synovial sheaths of the extensor tendons as well as in the joints themselves. There is a slight amount of grating to be felt on flexion of the knees. The child is otherwise healthy, the spleen cannot be felt, and there is no general enlargement of the lymphatic glands, though the cervical glands are palpable. No history of any previous disease could be obtained, and there are no signs of congenital syphilis: no local source of septic infection is present.

The diagnosis rests between joint trouble due to congenital syphilis and osteo-arthritis. There is, however, no history and no sign of the former. In favour of the latter diagnosis are the thickened synovial fringes, enlargement of the ends of the bones and grating of the knee-joints, and the appearance of the inter-phalangeal joints.

(*Exhibited February the* 21*st*, 1908.)

Discussion.

Mr. H. S. Clogg thought the joints might be tuberculous. There was no evidence of syphilis, and rheumatoid arthritis in a child generally started acutely, with pain and temperature, which the present child had never had. There was effusion in the knees, and in the wrist there seemed to be some affection of the tendon sheaths.

Dr. Porter Parkinson asked whether Calmette's reaction had been tried. There had been certain cases in which that reaction had not succeeded, and he recently had a peculiar case of typhoid fever in which the reaction came off very well, yet there was no evidence of tuberculosis in the case.

Dr. T. R. Whipham said he thought the case was one of so-called osteo-arthritis in children, and he differed from Mr. Clogg with regard to such cases, as he thought they occurred rapidly, painlessly, and without temperature. He asked whether skiagrams had been taken. One or two had been shown before the Society in which the lesions had proved to be peri-articular rather than in the bones themselves. In the present case he thought that the heads of the tibiæ were thick, as if there was some lipping of the bones, and it would be interesting to know if such was really the case. He also asked how long it was since the symptoms began.

Dr. George Carpenter regarded it as a case of osteo-arthritis in a child, and he agreed with Dr. Whipham's remarks on the subject. There was peri-articular thickening in the fingers, and the late Dr. Sansom showed a case which was very similar. In vol. i of the 'Reports' would be found skiagrams of the condition.

Dr. F. J. Poynton thought osteo-arthritis was not a very good name, because in that type there was not as a rule osteo-arthritis. It was very much like the cases described by Dr. Still in distribution and appearance. He asked whether it would be possible to extract some fluid from a joint and inject it into a guinea-pig.

Dr. F. Parkes Weber said that Mr. Turner's patient might be an example of the rheumatoid condition in children sometimes called osteo-arthritis. There was in the present case no enlargement of the spleen and practically no enlargement of lymphatic glands. He was, however, not inclined to exclude congenital syphilis, and asked whether anti-syphilitic treatment had been given a fair trial.

The Chairman (Dr. Edmund Cautley) agreed with the diagnosis of osteo-arthritis, though that was a bad term. Rheumatoid arthritis, or, better still, rheumatoid peri-arthritis, did not commit one to a definite idea of its pathology. But the case started peculiarly. That condition, in children and young adults, both in the chronic and the acute form, almost invariably began in the small joints of the hands. In the present instance it seemed to have begun in one knee. In such cases one should be on the look-out for some source of toxæmia, and attend particularly to the digestion and the excretions. In women it was possibly dependent on some leucorrhœal condition. Many different toxins might cause the disease.

58. HÆMOPHILIA WITH ADHESIONS IN THE KNEE-JOINT.

By KENNETH KELLIE, M.B.

The patient, a boy, aged 13 years, is one of a family of nine. The parents are healthy. Five of the children died in infancy. Of the other three the eldest son, aged thirty, has always suffered from bleeding. A daughter (married) is healthy, and a son healthy.

There is no history of bleeding in the collateral branches of the family.

The patient appeared quite healthy until three years of age, when he cut his finger, the bleeding of which was difficult to stop. Since then he has suffered from "bruises," which appear without any cause.

At the age of five his right knee became swollen and painful; this was followed shortly after by the left elbow becoming swollen.

Since then there have been many recurrences in these two joints.

Two years ago he woke up in the night with pain in the right knee and went into the Camberwell Infirmary, where he stayed until last August. During this time he had a bad hæmorrhage from a tooth.

When I first saw the patient the right knee was in the condition it is now—semi-flexed and with great limitation of movement.

A fortnight ago he had another swelling in the left arm. On examination there was a doughy swelling about the size of a crown just below the elbow-joint, which was slightly tender and painful on pronation.

I have brought the case before the Society in order to hear any opinions as to the treatment of the knee.

(*Exhibited February the 21st,* 1908.)

Mr. Lockhart Mummery thought the deformity was not so much a question of hæmophilia as the result of treatment. With effusion into the knee-joint the child was no doubt allowed to lie in bed with the knees drawn up, probably for many weeks, and no notice was taken of the fact that the knee was becoming ankylosed in that condition. The patient was suffering from adhesions and non-use of an inflamed joint, which had been allowed to bend. An anæsthetic should be given and an attempt made to break down the adhesions, though there was the risk by so doing of re-starting hæmorrhage into the joint. The coagulation time of the blood should be tested, then large doses of calcium chloride given for a week, and then tested again. If it became normal the attempt to break down the adhesions should be made. Otherwise he would leave it alone.

Dr. T. R. Whipham said the case was now a surgical one, and he asked whether any of the surgeons present had any experience of fibrolysin, and, if so, whether they thought it would be suitable for this case, injected subcutaneously.

Mr. Sydney Stephenson said that if fibrolysin were employed, as suggested, it must be introduced under the skin, and there would be the danger in such a case of hæmorrhages from the needle-punctures. He had had some little experience of its use in opacities of the cornea.

Dr. F. J. Poynton thought that if the adhesions were broken down now there would be no danger of bleeding to death into the joint, and the limb could be then got into position. Even if there were hæmorrhage into the joint it would be a better joint than now. Calcium chloride had been, in his hands, very disappointing, and he regarded the old remedy, cod-liver oil and iron, as the best for the condition.

Dr. Porter Parkinson agreed with Dr. Poynton as to the uselessness of calcium chloride. He had had cases in which new bruises had come out while the calcium chloride treatment was being carried out, and in another case hæmaturia appeared for apparently no other cause than the nurse turning the boy over in bed. The joint was a dangerous part to put fibrolysin into. He had treated one case with it as an experiment—a boy with a large scar in the neck made by a burn. It was contracting. Three injections per fortnight were made for three months, and then the boy got tired of it and left, but during that time there was considerable alteration in the scar. It first swelled up and got softer, and then the edge went back and the scar was diminishing. He thought its effect on the adhesions in the present case would not be great.

59 A CASE OF GRANULAR KIDNEY IN A GIRL, AGED FIFTEEN YEARS.

By CHARLES W. CHAPMAN, M.D.

A. E. W—, aged 15 years was admitted into the Hospital for Diseases of the Heart, Soho Square, on October the 15th, 1905. She had always been delicate, but her parents did not trouble about her health until three weeks previously, when advice was sought on account of extreme difficulty in breathing and swelling of the feet and legs.

Previous history.—Snuffles as a baby, pertussis at one year, and pleurisy at twelve years.

Present condition.—The nose is much depressed and has the appearance of that seen in old syphilitics. The child's development is that of one six years her junior, while the face has an aged aspect. The patient complained of severe asthmatic attacks which are worse at night, palpitation and general weakness.

Family history.—Both parents are living and in fair health. An uncle on the father's side died of phthisis. The mother has had two children born at term, of which the patient is one, and the other who died of marasmus at eight months. There were four miscarriages.

On examination the face was pale and there was puffiness under the eyes, and there was visible carotid pulsation. The cardiac dulness began at the lower part of the second space; the apex was in the sixth space, nipple line; the lateral dulness was from the mid-sternal line to 4 cm. external to the left nipple line. The impulse was heaving at the apex while at the base a tick-tack sensation was felt on palpation; the action was cantering in character. No thrill or murmur could be detected, but the second sound at the base was at times accentuated. Pulse 104, hard, small, regular; the radial arteries were hard and rolled under the finger, the brachials and carotid were thickened. There were a few small crepitations at the base of the left lung, back and front. The liver was enlarged nearly to the umbilicus. Urine acid, sp. gr. 1010, and contained $2\frac{1}{2}$ per cent. of albumin. Temperature 103° F. Mr. Eastes' report on the urine was: "The deposit is slight, but contains a fair number of tube casts, the majority of which are coarsely granular, and so broad as to suggest an origin in tubules which are denuded of epithelium. There are a few renal cells and red blood-discs free in the deposit."

By December the 14th the patient's condition had materially improved, but ten days later she vomited 8 oz. of blood. On January the 14th the respirations were 40 to the minute, for which no cause was discovered. The action of the heart was by this time persistently cantering and the second pulmonic accentuated; no murmur. Urine contained $4\frac{1}{2}$ per cent. of albumin, granular casts and red blood-discs. The dyspnœa was extreme at times and was relieved by strychnine and nitro-glycerine: some help was obtained by dry cupping over the kidneys. Dropsy began to increase so that the belly had to be tapped on two occasions; the face became almost square from œdema. The albuminuria increased until it reached 10 per cent. On May the 19th there were convulsions, and on the 31st the patient died.

At a post-mortem on the following day the heart, especially the left ventricle, was found to be hypertrophied and the cavities dilated: it weighed 12 oz., the muscle was pale and the valves competent. There was no atheroma. The liver weighed 38 oz. and was pale; spleen weighed 6 oz. Both kidneys were markedly granular; the right weighed 2 oz., the left $1\frac{1}{2}$ oz. The arteries were generally thickened. There was no evidence of the alleged pleurisy.

It is to be noted that the cardiac symptoms were so much in evidence when the child first came under medical care that it is not surprising the renal condition was overlooked. Moreover, there was no history of any one of the generally recognised causes of renal inflammation. I would suggest inherited syphilis as the most probable cause of the condition found in this patient. In support of this theory we have the history of snuffles in infancy and the state of the nose referred to, the retarded development, and prematurely old appearance. The mother had four miscarriages, while of the two born at term one died at eight months of marasmus.

With regard to treatment there was great relief of the cardiac pain and breathlessness from the exhibition of iodide of sodium, though it was not expected that the drug would have any effect upon the fibrosis.

Cases of syphilitic endarteritis and nephritis in infancy have been shown at this Society by Drs. H. Ashby, G. A. Sutherland and Mr. Thompson Walker, while Dr. George Carpenter has in the present year not only shown a case, but also contributed an exhaustive paper on "Nephritis in Infants" to the Society. See vols. i, iii and vii of 'The Reports of The Society for the Study of Disease in Children.'

In the present case it would appear that the cardiovascular changes, sometimes occurring in syphilitic infants, were delayed until a later period of life.

(*Exhibited March the 20th,* 1908.)

Discussion.

Dr. Porter Parkinson said the Society was much indebted to Dr. Chapman for his interesting paper. The cases of nephritis suspected to be due to syphilis were very interesting, and he hoped that at some time they would be capable of more direct proof. It had been long suspected that many forms of nephritis were due to syphilis, both congenital and acquired, and to the former even in the adult. As Dr. Bradford had recently said, a large number of cases of nephritis suspected to be due to cold were probably predisposed to by syphilis. Is was hoped that the organism of syphilis might be found in the kidney or urine in some of the cases.

60. AN UNUSUAL CASE OF ACUTE LEUCOCYTIC PLEURO-PNEUMONIA, WITH EXTENSIVE FIBRINOUS PLUGS VISIBLE TO THE NAKED EYE IN ENLARGED LYMPHATICS.

By GEORGE CARPENTER, M.D.

Susannah O—, aged 9 months, was admitted into the Queen's Hospital for Children on February the 1st, 1908, with the statement that she had been quite well until the day previous, when she retched but was not sick. Sickness, however, appeared the following day, the date on which she came into hospital. She was delicate at birth but lately had been stronger. The family history was satisfactory.

When admitted into hospital she was a fine, chubby child, with rapid respiration (64 to the minute) and a pulse-rate of 158 to the minute. The right apex was dull to percussion both back and front, and there was loud tubular breathing over this area and bronchophony; the breath-sounds elsewhere were vesicular. The temperature was 105° F.

The following day, although the temperature had fallen to 103° F., she was desperately ill. At 10 a.m. she became collapsed and she died the same afternoon.

Post-mortem.—Heart normal, patent foramen ovale; a little fluid in the pericardium. The spleen was somewhat enlarged, soft and congested; all the organs were free from tubercle.

Lungs.—The right upper lobe was solid and was covered with a thick deposit of yellowish-white lymph; part of the solid lung was red and part was pale. But the most striking feature was numerous yellowish-white areas scattered over the cut surface, looking much like follicular stomatitis. The accompanying drawings which I made of the condition at the time give a very good idea of the naked-eye appearances. The piece of tissue marked Fig. 45 was pale in colour, the portion marked Fig. 44

was red. Looked at more closely the yellowish-white areas presented a darker central part, which was a little depressed. Some of the lesions were finely branched, the areas were raised a trifle, and if they were seized and pulled they left the tissue intact, in which they apparently ramified branch-like. If pulled too vigorously they broke off and exposed little pits without any obvious walls, often pear-shaped depressions in the solid lung. When looked at more closely these pits or depressions in which the branch-like plugs were seated displayed *very* thin opaque walls.

Fig. 44.

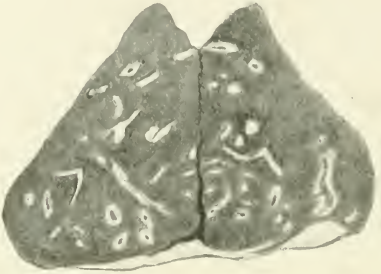
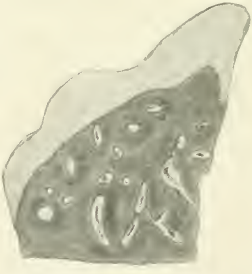


Fig. 45.



The condition was diagnosed as tubercular by one of the residents who made the autopsy, and the examination had been concluded when the specimen was accidentally brought to my notice.

Under the microscope.—Under a low power (Fig. 46 [× 10]) the solid lung is seen to be irregularly covered with considerable areas assuming a variety of shapes, and which evidently are not lung tissue proper. In a stained section they are inclined to be pale in the centre and to deeply stain in the periphery. They occur in the septal tissue as well as in the lung proper, and here and there they are seen to be in connection with the inflamed pleura.

Under a higher power these areas are found to be fibrinous with white corpuscles in the meshes of the fibrin.

In some parts they appear to be chiefly or wholly cellular, and in others the fibrin predominates. They are contained in channels, the thickness of whose walls is merely that of the endothelium of which they are constructed. In a few of the plugs a red blood-corpuscle can be seen but only here and there. These lymph-plugged channels appear to me to be dilated lymphatics which have become invaded by the same material as that which covers the inflamed pleura. The endothelial walls show no signs of proliferation or of inflammation.

The lung itself is consolidated and the alveoli are filled with cellular contents. In a few places fibrin can be seen along with the cells, but the predominant change is cellular—leucocytal mainly.

Some of the bronchi contain cellular plugs similar to those within the alveoli. Other bronchi have shed their epithelium, which is seen curled up in the lumen of the tube along with the cellular elements. Fibrin is seen, but seldom. In a few places there appears to be a slight exudate between the desquamated epithelium and the basement membrane, but this is unusual. The walls of the bronchi in some cases are infiltrated with cells. Nothing abnormal can be seen in the arteries. Some of the dilated lymphatics are closely associated with them, but in no case is envelopment complete. The sections

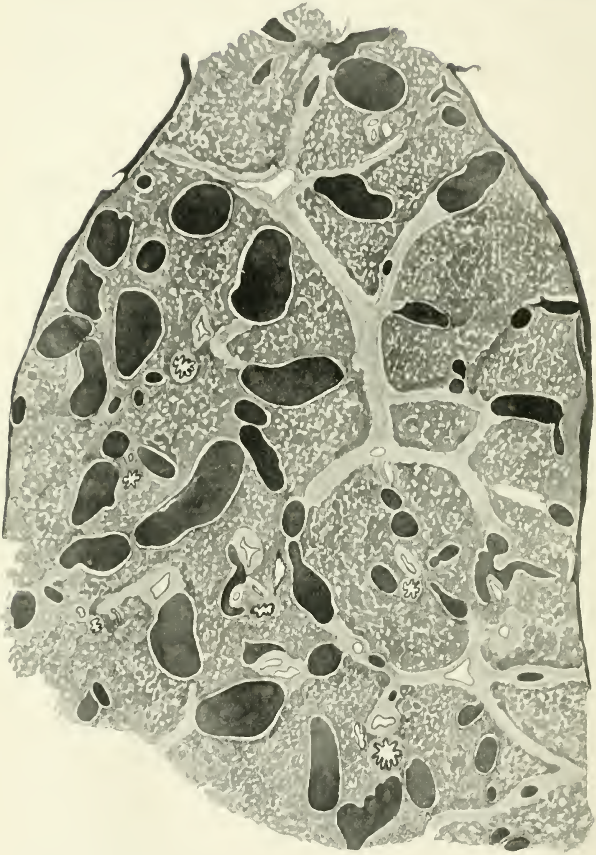


Fig. 46.—Section of pneumonic lung × 10 showing: the light coloured branch-like areas are the septa; the dark-stained areas are fibrinous plugs in dilated lymphatics, the leucocytes enmeshed taking the stain well, and the dark borders are the inflamed pleura composed of similar material. Note the bronchi and blood-vessels in the section.

stained by Gram show numerous capsuled diplococci, which occur in the lung proper, the plugs in the lymphatics and in the exudation on the pleura. A section stained for tubercle bacilli yielded negative results.

At the time of the autopsy the lymphatic origin of the plugs did not occur to me. Consequently I did not carefully examine the healthy lung for dilated lymphatics. Had they been dilated they would have surely shown irregularly-shaped pits and depressions, and they ought not to have escaped detection. I can well believe also that this condition might be readily mistaken for bronchiectasis to naked eye inspection. At the time I thought how like the lesions were to diphtheritic membranous casts of the bronchi, but the absence of a bronchial wall threw doubt upon that idea, though the subsequent detection of a very fine white boundary revived the original thought. My impression at that time was that the condition was a fibrinous extension up the bronchial tubes of an ordinary fibrinous pneumonia, but as I have shown my impression was wrong.

(*Exhibited March the 20th*, 1908.)

Discussion.

Dr. William Ewart said the naked-eye aspect, looking at the slide, gave him the impression that this was probably a case of peri-bronchitis with pneumonic consolidation; and that view seemed to be confirmed on microscopic inspection. There was a good deal of fibrin helping to consolidate the tissues, and there was an ocean of leucocytes proliferating all round in the peribronchial sheath and the peri-arterial sheath and in the lymphatics and compressing the alveoli. None of the bronchi were dilated. Many of them could be shown to be clear of any deposit, and lined with epithelium, which in places was squeezed somewhat laterally. Some of the bronchi were broken into by an invasion of leucocytic elements from outside. It seemed to be an unique specimen of a condition which might not be uncommon at the third day of a very acute attack. But generally the children died at a later stage, when the detachment of the necrotic fibrin had occurred, and there remained only then the excavations. The specimen might be regarded as illustrating the missing link in the evolution of the pathological condition of which Dr. Sharkey had given such a beautiful illustration—that of the "honeycombed lung." His original description of the condition was "acute bronchiectasis in children." There was certainly no bronchiolectasis in the present case, but simply a number of vacuoles of various sizes due to ulceration.

Dr. F. Parkes Weber, referring to Dr. Ewart's remarks, said he understood that the so-called "honeycomb-lung" was a condition affecting the whole of the lung or lungs, but in the present case the condition seemed to be quite localised to the apical region of one lung.

Dr. William Ewart, in reply to Dr. Weber, admitted that this was a fair criticism. It was improbable that a lung should be entirely invaded with pneumonia. Still, broncho-pneumonia often occurred scattered throughout a lung. There was, of course, a "honeycombed" lung of the cystic kind, but that was a totally different condition, more akin to a malformation. "Broncho-pneumonia" presented several varieties, and was a name really applicable to a group of diseases which required to be analysed and separately described. The peculiarities described by Dr. Carpenter in this specimen were a sufficient proof of that fact. Whether more strictly "pleuro-pneumonic" or "broncho-pneumonic," it belonged to the ulcerative or necrotic group; and, owing to the rapidity of the fatal ending, it happened to demonstrate a mode of production of the "honeycomb" condition which probably resembles in the main, though not in every detail, that which may occur in other members of the group.

Dr. George Carpenter, in reply, said that some of the bronchi were normal, some contained cellular plugs, in others the epithelium was desquamated, and in others there was some cellular infiltration. The specimen was an example of ordinary pleuro-pneumonia of leucocytal type. But the interest in the case lay, not in the pleuro-pneumonia, but the very dilated lymphatic vessels. The fibrin plugs in these were identical in structure with the exudate on the pleura. The endothelial walls of the dilated lymphatics showed no signs of proliferation. He had not seen a similar case, and he had hoped to hear numerous opinions upon it. He did not think the microscopical features were in the smallest degree suggestive of vacuoles of various sizes due to ulceration; on the contrary. The fibrin plugs were not necrotic, and their cellular contents were demonstrably "living," and products of the blood and these plugs were contained within spaces possessing an obvious endothelial lining, and in situations occupied by the lymphatics, and they were without a suspicion of a melting away of contiguous structures, such as could occur in any such process and that which had been suggested.

61. CASE OF WIDESPREAD INFANTILE PARALYSIS, WITH AFFECTION OF THE ABDOMINAL MUSCLE AND CONSEQUENT BALLOONING.

By F. LANGMEAD, M.D.

History.—W. B—, male, aged 1 year and 8 months. At three months the patient is said to have had bronchopneumonia, and afterwards it was noticed that both legs and arms were motionless, and that the child could move neither head nor body. The arms rapidly got well, and after three months seemed to have become normal, and at the same time he could again move and support his head. He was first seen at Paddington Green Children's Hospital in May, 1907, eight months after the onset, and had had no treatment since the acute illness. The legs were now seen to be flaccid, cold and blue; they could just be moved below the knees, but the thighs remained motionless. Very slight movement in the toes was possible. He was unable to sit up; knee-jerks lost. Since then, as result of massage and passive movements, there has been gradual improvement, but there is still very widespread paralysis. He can now sit up by rolling over on to his right side, and is quite steady in the sitting posture. Both legs are still flaccid, paralysed and atrophied, the left more than the right. They are kept abducted and externally rotated, or, if sitting, are drawn up and kept in the tailor position. There is double talipes equino-varus, the tendo Achillis tendons being rigidly contracted. He can both flex and extend the legs and move the toes on each side. He can hold the right leg rigid, and flex it by the quadratus and psoas muscles on the right side, but not on the left. Hypermobility of the hips allows of meeting of the soles of the feet behind the neck. Both knee-jerks have returned, the right more than the left. Plantar responses present and flexor. He makes no attempt to stand. A marked scoliosis has developed, the convexity being to the left in the lumbar and lower dorsal spine.

The abdominal muscles have been seriously affected, the oblique and transversalis apparently more than the recti, and the left side more than the right.

Last August it was first noticed that when the child coughed or cried two rounded tumours projected on the left side. Each was about the size of a large orange, and was evidently the result of a local yielding of the abdominal parietes. One was situated in front, just above and to the left of the umbilicus, the other behind, just below the ribs in the left lumbar region. These swellings have now become less definite, but even now their outlines can be clearly defined if the child cries or coughs vigorously. Through these gaps in the abdominal wall the spleen and left kidney may be readily felt.

There is no evidence of diaphragmatic or intercostal paralysis. There is incontinence of urine and fæces. The interest of the case lies chiefly in (1) the extensive affection of the anterior cornua, which at first must have included both the cervical and lumbar enlargements and a considerable part of the intervening portion of the cord, and in (2) the local paralysis of abdominal muscles, giving rise to ballooning, like that which sometimes occurs as a congenital malformation.

(*Exhibited March the 20th,* 1908.)

Discussion.

Dr. F. Parkes Weber said an elaborate study of the subject had recently been made by Professor Roman von Baracz, of Lemberg,* and he thought it was the first monograph to draw attention to the clinical group of cases in which there was a "spurious ventral hernia" as a result of acute anterior poliomyelitis affecting the muscles of the abdominal wall. It was suggested that there might be rare cases of acute anterior poliomyelitis in which only a portion of the abdominal wall was affected, so as to give rise to spurious ventral hernia without there being any (at least any permanent) evidence of the limbs and other parts of the body having been paralysed. He asked whether there had been any work published in England which referred to the existence of such cases.

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* See the review by R. Romme on "La Poliomyélite et les Pseudo-Hernies Ventrales" (that is, on "Poliomyelitis and Abdominal Ballooning"), 'Presse Médicale,' Paris, February the 22nd, 1908, p. 124. Dr. Weber wonders whether any of the cases of abdominal ballooning due to local congenital deficiency in the abdominal wall, such as those mentioned by Dr. G. A. Sutherland, may be originally caused by intra-uterine poliomyelitis with resulting imperfect development in the affected muscles.

Dr. Edmund Cautley said that in the Medical Society's 'Transactions' cases of the kind were recorded, *i.e.* where there was no other evidence of acute anterior poliomyelitis than the ballooning of the abdominal wall, and yet at the time these cases were recognised as probably due to that disease. One such case had come under his care about seven years ago.

Dr. G. A. Sutherland said that when the subject was discussed on a former occasion he said the complication was very rare in his experience, but others said that such an impression was due to its not having been looked for in cases of infantile paralysis, as both back and abdominal muscles were frequently paralysed in the disease. That might be during the acute stage, but it was not common, clinically, to observe such an effect in the trunk muscles later on. The first thing that struck him was the very early onset in Dr. Langmead's case, as the infant was only three months old, and that increased the difficulty of diagnosis. He had seen one patient at a comparatively early stage of the disease, and when the child strained the whole right side of the abdomen bulged markedly, so that the lower part hung over Poupart's ligament. Improvement went on, and the ultimate condition came to be a localised paralysis of the muscles in the right hypochondrium. A condition which might closely simulate infantile paralysis was a congenital absence of a certain area of muscular tissue in the abdominal wall. He showed a photograph of a case of congenital absence of muscle in the abdominal wall on the left side, producing localised ballooning on straining. Dr. Langmead's case showed the selective action of the poison in infantile paralysis. He thought he felt a thinning of the part at which the bulging occurred, not in one muscle in its entire extent, but parts of muscles. The same condition occurred in the limbs, but it was shown more strikingly in the abdominal muscles. The amount of spinal curvature which developed afterwards in cases of abdominal paralysis was often out of proportion to the extent of the lesion. In the present case that might not become so marked, because probably the child would never be able to walk.

The Chairman (Dr. C. W. Chapman) said he had seen a child born with distinct absence of all abdominal covering above the umbilicus, and the bowel protruded completely.

62 A MONGOLIAN IMBECILE, SHOWING OTHER CONGENITAL MALFORMATIONS.

By F. LANGMEAD, M.D.

Leonard G—, aged 6 months. The patient is the first child. There were no miscarriages. There is no history of malformation in the family.

He is a typical example of a Mongolian imbecile. The eyes slant, the tongue is pointed and frequently protruding, the face is flat and constantly grimacing. There is a slight umbilical hernia. The back of the head is, however, not flat, nor is the little finger characteristically incurved. There are no lingual fissures. No blue patches on the buttocks.

Many authors have pointed out the frequency with which this form of imbecility is associated with congenital malformations.

In this case, excluding the umbilical hernia and a deep post-anal dimple, there are two:

(*a*) A congenitally malformed heart. A loud systolic bruit can be heard at the left side of the lower end of the sternum, and at the apex, probably due to an imperfect septum ventriculorum.

(*b*) An accessory auricle on the right side.

(*Exhibited March the 20th*, 1908.)

Discussion.

The Chairman (Dr. C. W. Chapman) said he had wondered whether such cases were now more frequent than formerly, and if so, whether they were connected with the attempts made at abortion by women to whom maternity was an objection. Three years ago a woman in good position wrote asking for his assistance in getting rid of a child she was carrying. She had had two or three children, and was very fond of hunting. He talked to her very strongly on the matter, but he believed she persisted in her efforts. That child was a Mongolian idiot. The other children were absolutely healthy and both parents were very intelligent. He asked whether any member's experience agreed with that.

Mr. Sydney Stephenson said that no practical person would attach undue importance to squint in a baby aged 6 months, but there was a squint in the left eye of this case, and his opinion, from an imperfect examination, was that it was produced by a congenital defect of abduction, not only in the left eye but also in the right. If so, it was interesting, as it fell into line with the other malformations which had been described by Dr. Langmead.

Dr. G. E. Shuttleworth asked whether there was any history of syphilis in the parents, or any signs of the disease in the child. There was no doubt the case was one presenting "Mongolian" traits, the most characteristic evidence in this instance being the facial physiognomy rather than the conformation of the skull. The present case had more occipital development than one usually found in a Mongolian idiot. The tongue was large and coarsely papillated, though not fissured, and it had been remarked by Dr. John Thomson, of Edinburgh, that the tongue was not usually furrowed before four years of age. It would be interesting to learn in a few months' time how the intelligence progressed. This was the first-born child of a young but apparently delicate mother after four years of married life, whereas the majority of such cases seemed to have been begotten in the declining years of sexual capacity. In regard to the remark of the Chairman as to the influence in producing Mongolism of noxious drugs taken to bring about abortion, he could not remember a clear case of that, but some time ago, in ascertaining the ætiology of a Mongolian child, the daughter of a lady from Holland, who had a contracted pelvis, he found that a Dutch lady doctor had persuaded her to undergo a starvation diet so as to have a small viable child. The result was the birth of a Mongolian imbecile at full term.

Dr. G. A. Sutherland said there was an affection which neurologists were calling attention to, namely, amyotonia congenita, but he did not know whether it was a distinct pathological entity. No better example of amyotonia could be found than was shown by Mongolian infants.

Dr. F. Langmead said there did not appear to have been syphilis in either parents or child. In conjunction with Dr. Garrod he had published a case of Mongolian imbecility in a child in whom there were many more congenital malformations than in the present case. There were complete transposition of viscera, very marked congenital malformation of the heart, and imperforate anus.

63. A CASE OF CONGENITAL MORBUS CORDIS IN A MONGOL.

By GEORGE CARPENTER, M.D.

Minnie L—, aged 5 months, a Mongol, was admitted into the Queen's Hospital on March the 4th, 1908. A breast-fed child, she was apparently healthy until three days before, when she became sick and declined her food. The mother thinks it changed to a blue colour soon after birth and did not thrive well. When admitted into hospital she was blue and there were signs of bronchitis, but the former disappeared, though occasionally she has attacks of cyanosis now.

Heart.—The cardiac impulse is in the sixth interspace half a finger outside the nipple line. The nipple is on the fourth rib. The heart is outlined above at the third costal cartilages and the corresponding portion of the sternum. Its extreme right border is a finger's breadth to the right of the right sternal margin. On the left it reaches the nipple, which is two fingers' breadths to the left of the left sternal margin, and from thence extends obliquely downwards to the apex beat.

There is a loud systolic murmur. This is best heard at a point midway between the apex beat and the junction of the seventh costal cartilage with the sternum, next over this spot, next over the apex beat, much less distinctly over the third left interspace near the sternum, next just outside the left nipple, and next over the right nipple but somewhat higher.

Over the top of the sternum it is audible and it can also just be heard in the neck, but it is not loud there. It is heard in the back faintly, the left side the louder of the two. There is no thrill. A valvular snap is felt all over the præcordium with the systole.

Hæmoglobin, 70 per cent.; red blood-corpuscles, 4,800,000; white blood-corpuscles, 15,000; differential count: polymorphs, 65 per cent.; lymphocytes, 30 per cent.; mononuclears, 3 per cent.; eosinophiles, 2 per cent.

The exhibitor said he was not inclined to express any opinion as to the nature of the malformation.

On March the 11th there were a few consonating râles to be heard over the left scapular angle, and on the 16th there was diminished resonance over the left lung behind, just below the angle of the scapula, with tubular breathing and consonating râles. Over the right lung behind there were a few bubbling râles to be heard over both lobes.

(*Exhibited March the 20th*, 1908.)

Discussion.

Dr. Parkes Weber remarked that pathologists and clinicians were anxious to find whether there was any post-mortem evidence of excessive activity in the formation of red corpuscles in the bone-marrow in cases of polycythæmia with congenital heart-disease. It was really astonishing that scarcely any accurate investigations regarding that point had as yet been recorded, that is to say, by careful macroscopic and microscopic examination of the bone-marrow after death.

Dr. F. W. Higgs said he thought one ought to attempt to diagnose such cases, and his view was that there was stenosis of the pulmonary artery and patency of the ventricular septum. He thought the heart murmur conducted down the sternum and to the apex was indicative of the latter. On the right side of the sternum a murmur of different pitch was heard, probably produced in the pulmonary artery. This was supported by the history that the child had attacks of cyanosis. Cases in which both lesions were present were apt to go on longer than others, as the patent septum ventriculi acted as a safety valve in dilatation of the right ventricle caused by pulmonary stenosis.

Dr. George Carpenter, in reply, said amyotonia was very marked in the case he exhibited. The hæmoglobin was 70 per cent., the red corpuscles 4,800,000, the white 15,000 per c.mm. Therefore the reds seemed to be quite normal, or even in excess of it. In congenital malformation of the heart there was not excess of reds unless the child was blue. There was a suggestion that the red corpuscles in cyanosis were manufactured in the bone-marrow. He had not been able to verify that statement, but he would take the first opportunity of doing so. In answer to Dr. Higgs, he thought it impossible to make a diagnosis in infants, and he, as a result of experience, declined to express in them any positive opinion, though Dr. Higgs' diagnosis might be correct; it was certainly one of the possibilities. When an attempt was made the post-mortem examination often revealed something entirely different to the supposed lesion, but the events occurring in such cases should be carefully recorded if we are to hope to make any progress in diagnosis.

Postscript.

For sequel see exhibit No. 83, p. 339.

64. A CASE OF CHRONIC RHEUMATOID DISEASE IN A CHILD WITH ANKYLOSIS OF BOTH KNEE JOINTS.

By F. PARKES WEBER, M.D.

The patient, Arthur S—, now aged 10½ years, was the first of the two cases described by me in a paper entitled "Still's Type of Chronic Joint Disease in Children and the so-called Tuberculous Rheumatism" ('British Journal of Children's Diseases,' May, 1905, p. 208). The disease, which seems to have commenced acutely in the latter part of 1900, was chiefly characterised by swelling of the hands and feet, cervical rigidity, and moderate enlargement of the spleen and superficial lymphatic glands, that is to say, whilst the patient was first under my care in the German Hospital (1901). Under treatment by rest, aspirin, iodide of iron, arsenic, malt, etc., the condition, in regard to the swelling and tenderness of joints and the general appearance of the child, fluctuated considerably. The wasting of the muscles of the limbs at first progressed. Occasionally there was moderate fever, and about the beginning of November, 1901, there was an attack of endocarditis, and the heart began to show signs of mitral valve disease. Since that time the heart has remained somewhat enlarged, though no cardiac murmur can at present be detected, excepting a slight systolic one of

Potain's "cardio-pulmonary" type, present only during inspiration. In February, 1902, the temperature began to remain below 100° F., and the child had a good appetite, put on flesh, and seemed happy and free from pain and tenderness in the joints. In fact the absence or relative absence of pain and tenderness in the swollen hands and feet and at the other affected articulations was specially noteworthy in this case, and seems to be remarkably characteristic of the whole clinical class of cases to which this one belongs. Röntgen ray photographs showed no bony changes at the articulations, the exudation being apparently gelatinous and periarticular.

In May, 1902, there was a slight attack of (catarrhal?) jaundice, and unfortunately in December of the same year the boy contracted enteric fever. In December, 1903, he was again in the hospital with occasional fever, swelling of the knees, and some increase in cervical stiffness. In spite of all this, however, very great improvement took place, and the following note describes the condition when I saw him in December, 1904: "He looks well and goes to school. There is scarcely any stiffness in the vertebral column and the movements in his limbs are quite good. There is still puffiness over the back of the proximal part of each metacarpus and some thickening of the fingers; also some thickening over the back of both feet and some swelling of both knee-joints. Apparently no pain anywhere. The spleen can just be felt on inspiration. The axillary glands on both sides are slightly enlarged. The cardiac apex-beat is half an inch outside the nipple line, and there is a slight systolic apical murmur."

When I saw him in August, 1906, his condition appeared satisfactory, though he had slight genu valgum. He looked bright and active and walked well. I thought that he was practically cured. However, I understand that later on in the same year he again fell ill at home, but I did not see him till August, 1907, when he was re-admitted under my care in a quite crippled condition.

There was then much swelling of the knees, elbows, ankles, wrists and sterno-clavicular joints, and less swelling of some of the smaller joints of the hands. The legs were drawn up, that is to say, the hip-joints and knee-joints were flexed, and owing to adhesions the knee-joints could not be extended. The legs had been apparently drawn up for about two months. There was a small recent bed-sore in the sacral region. The movements of the vertebral column and of the head were fairly good. The ribs showed very marked rickety "beading." There was scarcely any fever, and the joint trouble seemed to be practically unaccompanied by pain or tenderness.

In the hospital the boy's condition improved slowly, and on January the 7th, 1908, under chloroform-anæsthesia, both knee-joints were forcibly extended (Dr. E. Michels) and fixed in plaster bandages. During the requisite manipulation of the joints the upper tibial epiphyses were unavoidably separated on both sides. The plaster bandages were removed on January the 31st, but the left knee-joint soon tended to become again flexed, and consequently had to be put up again in plaster. He is now beginning to walk with help, but this is very difficult, not only on account of the fixation of the knee-joints, but also on account of great limitation of movement in the hip-joints. Owing to partial (incomplete) fibrous ankylosis of these joints in a faulty position, the thighs cannot be fully extended, and the boy cannot therefore stand up straight without considerable lordosis of the lower part of his spinal column. There is still a certain amount of swelling at the backs of both wrists and proximal metacarpal regions, and of some of the interphalangeal joints and of both elbows (about the head of the radius). The right knee-joint (the other one is at present in plaster) is slightly puffy, and so are both ankles and the dorsal regions of the feet. The ribs are "beaded." The spleen is evidently (by percussion) enlarged, though the edge cannot at present be felt. The liver is apparently not enlarged. The cardiac apex-beat is felt in the fifth intercostal space, half an inch outside the nipple line; the heart's action is forcible, but, as already stated, no murmur indicative of valvular disease can at present be detected. Of the superficial lymphatic glands, the axillary ones are those chiefly enlarged, but they are discrete, and none of them are larger than about the size of a hazel-nut. There is practically no stiffness of the neck or vertebral column. Examination of the blood shows the presence of slight anæmia. The urine is free from sugar and albumin. In the way of drugs the boy has been taking iodide of iron.

In February, 1908, Calmette's "ophthalmo-reaction" for tuberculosis (1 per cent. fluid as recommended by Calmette) gave a completely negative result, though von Pirquet's "cuti-reaction" for tuberculosis (vaccination on the forearm with Koch's old tuberculin, 25 per cent.) gave a positive result.

This positive "cuti-reaction" is interesting. In a girl, aged 12½ years, with chronic joint disease of somewhat similar type to that in the case of Arthur S—, I once obtained a positive general reaction by the subcutaneous injection of Koch's old tuberculin for diagnostic purposes.* After the injection of five milligrammes her temperature rose to 101° F., but the general febrile reaction appeared not to be accompanied by any local reaction in the joints, and it is quite possible that somewhere in this girl's body a latent tuberculous focus existed, which had no connection with the chronic joint disease. Anyhow, in Arthur S—, even if the positive "cuti-reaction" be accepted as indicative of the presence of some latent tuberculous lesion, I think it very unlikely that the arthritic trouble has any direct connection with it. I may mention that there certainly is nothing in the case to suggest a congenital syphilitic origin for the joint disease. That there is a rachitic element in the case must be admitted (the boy's ribs are at present markedly beaded, though he is 10½ years old), but whether the rickets can be causally connected with the polyarticular rheumatoid disease is doubtful. The development of the mitral valve lesion in 1901 may be taken as probably pointing to the presence at that time of actual rheumatism.* The articular affection, however, in the present case, as in other examples of Still's type of chronic joint disease in children, differs from true articular rheumatism by the relative absence of pain and tenderness, and by the chronic relapsing course of the disease.

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* See description of the case in the 'British Journal of Children's Diseases,' *loc. cit.* (second case described).

In connection with the present case it is also interesting to find that Rupert Waterhouse, in his investigations as to the state of the lymphatic glands in rheumatoid arthritis ('St. Bartholomew's Hospital Reports,' London, 1907, vol. xxx, p. 107), has come to the conclusion that enlargement, moderate in degree, of the superficial lymphatic glands is almost always present in polyarticular rheumatoid arthritis of the type that commonly attacks young women, when the affection has lasted more than a few months. He maintains that glandular enlargement can no longer be considered a distinctive feature of Still's type of joint disease in children.

(*Exhibited March the 20th*, 1908.)

Discussion.

Dr. Edmund Cautley said such cases were always difficult to diagnose. He asked whether there was any evidence of gonorrhœal infection—not necessarily by the penis—as the case seemed like one of gonorrhœal arthritis. Also was it not a fact that von Pirquet's reaction was valueless after two years of age? He had very little confidence in Calmette's serum reaction, which he had tried on many occasions.

Dr. F. Parkes Weber, in reply, said that when he first saw the case it did not at all resemble gonorrhœal rheumatism; it was painless, and the hands and fingers were not affected in the way common in adult patients with gonorrhœal rheumatism when it affected the hands. The swelling was chiefly just over the wrist and over the proximal part of the metacarpal bones on both sides. The fixation of the hip and knee-joints had only recently occurred. Dr. Weber thought that gonorrhœa could be absolutely excluded in regard to the ætiology of the articular affection in the present case, and probably, indeed, in all cases of Still's type of joint-disease in children.

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* F. J. Poynton has recently described the case of a boy, aged 4½ years, suffering from a chronic joint affection of a type similar to those described by Still, but associated with obvious valvular disease of the heart ('Edinburgh Medical Journal,' September, 1907, p. 232).

65. A CASE OF TUBERCULOSIS OF THE KIDNEY, WITH PYONEPHROSIS.

By J. PORTER PARKINSON, M.D.

Violet G—, aged 9 years, was admitted into the Queen's Hospital for Children for feverish attacks with no apparent cause.

She had suffered from no illness, except measles three years ago. There was no family history of phthisis.

The history was that during the last eighteen months she had suffered from attacks lasting a few days, during which she was feverish and complained of headache and pain in the left side of the abdomen. There was no vomiting or trouble with the bowels. During the last attack the mother noticed that a lot of custard-like material came away with the urine. The attacks were generally at first mistaken for typhoid fever.

On admission the temperature was normal. No abnormal signs were found in the heart or lungs. The abdomen was slightly tender on palpation, the liver was not enlarged, but the spleen was felt extending slightly below the costal margin, and immediately below this, but more deeply situated, was a distinct mass extending downwards for two or three inches; it was resistant and slightly nodular, but with a rounded lower end like a kidney, it was dull on percussion except for the colon resonance, which could be obtained over it. On the right side there was a sense of resistance below the liver suggesting enlargement of the right kidney. There was no enlargement of any of the external lymphatic glands, but *per rectum* two hard enlarged glands could be felt in the right iliac region. The urine was reduced in amount, being about 12 to 16 ounces daily; it was acid and deposited a large amount of pus; there was a trace of albumin, but no blood. The patient weighed 3 st. 7¾ lbs. and looked well nourished and healthy, though somewhat anæmic. There was no pain or frequency of micturition.

The urine was examined for tubercle bacilli, and after three examinations these were found. A skiagram revealed nothing abnormal.

Calmette's ophthalmo-tuberculin reaction was tried twice with negative results. During her five weeks in the hospital she has increased 5 lbs. in weight, and the pus has almost disappeared from the urine, but the kidney tumour has not altered in size or character.

The separated urine of the two kidneys has not yet been obtained, but I believe the left ureter is almost completely blocked and that the pus in the urine comes from the right kidney.

(*Exhibited March the 20th*, 1908.)

Discussion.

The Chairman (Dr. C. W. Chapman) commented on the fact that although there was so much renal trouble the child seemed well. It showed that too much reliance should not be placed on a healthy appearance.

Dr. Edmund Cautley asked whether Dr. Parkinson was sure that the bacilli found were not smegma bacilli, as they were often difficult to distinguish from tubercle bacilli, and were present in girls as well as boys. He suggested trying the tuberculin reaction, with a view to diagnosis and treatment.

Mr. Hugh Lett said that in these cases if only one kidney was affected, and the bladder was sound, it was best to do an exploratory operation, to find the extent of damage to the kidney, and resect that part, leaving the rest of the organ. But where both kidneys were involved, one of them to a considerable extent, the question of operation became an interesting one. One reason for operation was the fact that such cases subsequently became infected with the *Bacillus coli*, after which the patient went rapidly downhill. In this case an attempt should be made to find out the efficiency of the opposite kidney by separation of the urine, and if the other kidney was working well he would advise nephrectomy. Tuberculous affections of the kidney were very chronic, and he had seen cases which had existed for years, the patient having gone on well under palliative treatment, but the present case seemed to be one requiring operation.

Dr. George Carpenter said that Dr. Parkinson was fortunate in being able to discover tubercle bacilli in the urine and to make a diagnosis from that source. It was not always that the bacillus could be found in the urine, because the corresponding ureter might be blocked. That had happened in two cases under his care.

Thus, a girl (Amy F—), aged 4 years, was seen by him on June the 13th, 1895, and was brought by her mother for advice on account of wasting. Six months previous she had been treated at Westminster Hospital for diarrhœa, and after that she was sent by the parish of Camberwell to a convalescent home at Herne Bay. She always had been troubled with her water, so her mother said, having a frequent desire to micturate, but could only pass a little at a time. Three months before there was "slime" in it, but now it is "quite clear, like the white of egg." It smelt "strong." Her mother had never noticed the stomach to be large, but the child had complained of pain then—that is, twelve months before. Both parents were well and healthy, but the paternal grandmother suffered from asthma. When he saw her the labia were very swollen and pale. There was considerable enlargement of her abdomen, and the abdominal veins were very prominent. In the left renal region there was a large globular swelling. The rectum and descending colon were full of fæces. On removing the fæcal lumps the colon could be felt as a cord passing vertically over the face of the tumour. While the examination was being made the child passed water, and this was perfectly clear. Nothing abnormal was felt *per rectum* in the ureters. He made an exploratory puncture in the renal region behind and withdrew a syringeful of pus, which contained tubercle bacilli. The kidney was removed and the girl made an uninterrupted recovery. The accompanying sketch represents the appearance and size of the organ on its removal (Fig. 47).

Another child (Elizabeth McK—), aged 21 months, was brought to him for wasting and a bad cough on March the 20th, 1890. The child was weaned at ten months, and since then had been fed "on the same food as the family." The mother complained that "everything she ate passed through her immediately, and that she was always picking herself and was very thirsty."

She also passed "a lot of water." The water was thick and had a white deposit like milk. This condition of the urine coincided with the commencement of the wasting. When he saw her she was much wasted, had a sallow complexion, and appeared to be ill. She had molluscum contagiosum on her face. The anterior fontanelle admitted the tips of three fingers. On examination there was a large, firm, globular tumour occupying the left side of the abdomen. Below it extended as far as the iliac crest, and it reached to within three inches of the umbilicus. *Per rectum* the smooth globular lower end of the tumour could be felt in the iliac fossa. A sense of deep fluctuation could be obtained on

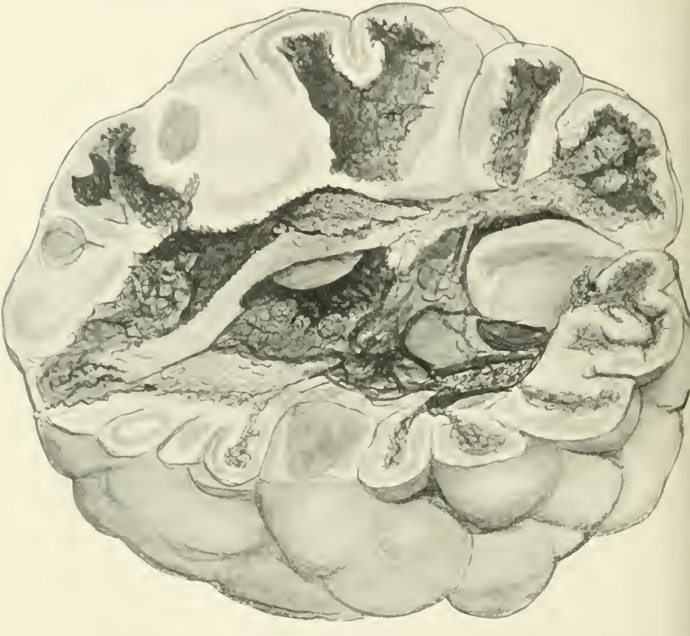


FIG. 47.—Kidney of the infantile type infiltrated with tubercle showing caseation (lightly-shaded areas of the cut surface) and excavation of the renal parenchyma. The excavated areas contained pus. From a child, aged 4 years. Actual size.

manipulation. The upper end of the tumour disappeared under the costal margin. The bladder and ureters felt normal. Lying on it was the spleen, which was a trifle enlarged. The free edge of the liver was a finger's breadth below the costal margin in the nipple line. There was no enlargement of the abdominal lymphatic glands or other lymph nodes. The child soon died, and a post-mortem examination was not consented to, but he made some observations by removing the parts *per rectum* with the following results: The pelvis of the kidney was very dilated and filled with pus. The left ureter was blocked by a phosphatic calculus the size of a small marble. The calices were much dilated and filled with pus. There were numerous tubercular abscesses in the kidney substance, and tubercle bacilli were demonstrated.

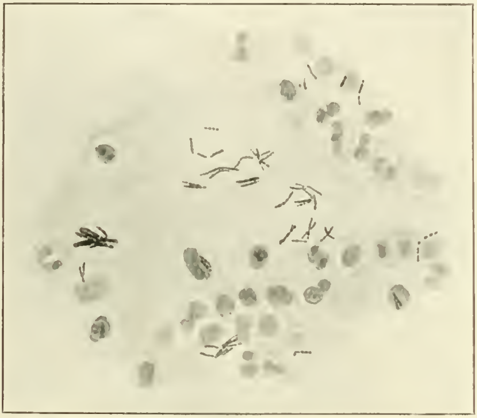


FIG. 48.—Tubercle bacilli in purulent urine.

He also exhibited a coloured sketch of tubercle bacilli from the urine of a third case which came under observation about the same time, a child with general tuberculosis of the genito-urinary organs, with purulent urine (Fig. 48).

If such cases were seen early, and there was no involvement of the bladder, the surgical removal of the kidney was satisfactory, because the condition often began on one side. But where ureters on both sides and the bladder were involved operation was out of the question.

Dr. Porter Parkinson, in reply, said that when he sent the specimen to the pathologist he asked that the alcohol decolorisation method should be used, and every precaution be taken to differentiate the tubercle bacillus from the smegma bacillus. The bacillus was not decolorised by alcohol, as the smegma bacillus usually was. He had thought of trying tuberculin, but as the child improved so much he thought it would do well without. He asked for the opsonic index to be taken, but he did not yet know what it was. He thought the case would be far better treated by ordinary medical measures than by surgery. He had seen cases treated surgically in which a fistula resulted, which was infected with bacilli, and the last state of the patient was worse than the first. He thought the left kidney in the present case was not now acting, as it was apparently a bag of pus.

66. SUBCUTANEOUS FIBROUS NODULES IN A NON-RHEUMATIC INFANT.

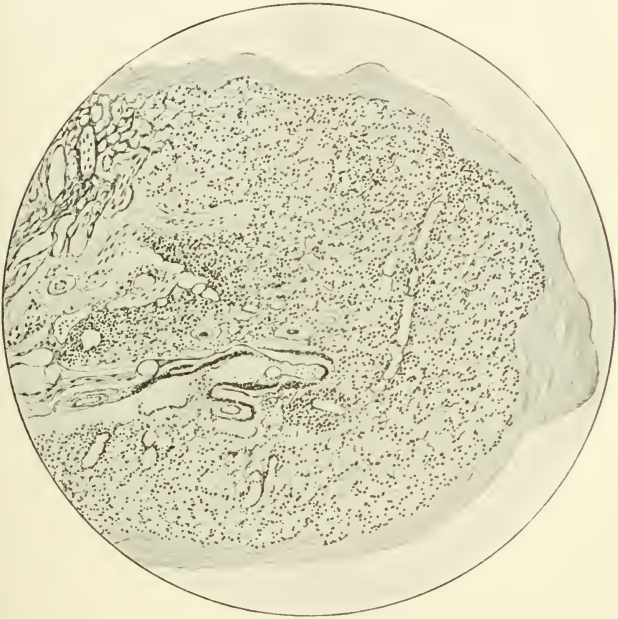
By GEORGE CARPENTER, M.D.

Elizabeth E—, aged 7 months, was admitted into the Queen's Hospital for Children on December the 31st, 1907. Four of her second cousins on the maternal side died of phthisis. She was breast-fed until three months old and since then had been reared on cow's milk and water. At four months old she suffered from diarrhœa and vomiting and had attended as an out-patient since then. She had wasted for three months, but had not been sick for seven weeks and was also free from diarrhœa.

On admission she was a very ill-nourished, pallid child, with marked desquamation of the head and trunk, and suffered from an opaque thin discharge from the nostrils. The liver was one finger's breath below the costal margin in the nipple line, but the spleen was not enlarged. The urine contained a trace of albumin and some pus; no rickets; no cranio-tabes; heart normal. The buttocks were excoriated.

Cutaneous hæmorrhages.—On the arms just by the axillæ there were two cutaneous hæmorrhages the size of the tip of the index finger, together with a small hæmorrhage over the right deltoid in front. On the abdomen on both sides were small cutaneous hæmorrhages, and on the left side of the lumbar spine were also two hæmorrhages the size of large peas. The glands in the groins were palpable, the size of a pea. The supratrochlear glands were not enlarged but the glands in the axillæ were palpable.

Fig. 49.



Subcutaneous nodules.—Just at the angle of the left scapula, of the size of a small pea, movable over a radius of $\frac{1}{4}$th inch, was a subcutaneous nodule. This was subsequently removed. Over the occiput were about a dozen nodules varying in size from a small shot to a small pea, and probably of the same nature. While in hospital there was no evidence of rheumatism. The subcutaneous nodule on the scapula was removed under an anaesthetic on January the 10th, 1908. On the 13th there was pus in the urine but no albumin. On the 14th she was taken from hospital by her mother.

Microscopically the growth consisted of an outside fibrous capsule containing but few nuclei, and sending fibrous trabeculæ into the interior. In the interstices of these was a very delicate areolar tissue richly supplied with cells. No micro-organisms were discovered.

(*Exhibited March the 20th*, 1908.)

67. CASE OF LARYNGEAL STRIDOR IN AN INFANT.

By GEORGE CARPENTER, M.D.

John F—, aged 6 months, was brought to the Queen's Hospital for Children January the 23rd, 1908, because he "made a noise like croup."

He has made the same noise since two weeks after birth, but is said to become better if he gets warm by the fire. If he has a cold stridor becomes worse. Sometimes it stops during the night, at others it goes on during his sleep.

On admission he was a fat baby, snuffling loudly, with muco-pus appearing at his anterior nares, but the bulk of the discharge found its exit by the posterior nares, and his mouth was seen to be full of muco-pus. He had loud snoring laryngeal stridor with sucking-in of the lower ribs and supra-clavicular regions. The stridor was associated with a natural laryngeal cry which demonstrated that the vocal cords were intact. A digital examination of the glottis did not lead to the detection of any abnormalities, and the posterior nares could not be felt. It was found to be impossible to view the part with a laryngeal mirror

—the mouth very quickly filled with muco-pus which could not be cleared away. The spleen was one finger's breadth below the costal margin and the liver could be percussed downwards to the same extent. The ribs were beaded, not much, but the bossing was appreciable. There was no cranio-tabes. The buttocks were excoriated. The blood was examined for spirochætes but with a negative result.

Hæmoglobin, 50 per cent.; red blood-corpuscles, 3,200,000 per c.mm; white blood-corpuscles, 13,500 per c.mm. Differential count, normal proportions.

Snoring was audible all over the chest, the sound being conducted from the larynx. There was kyphosis in the dorso-lumbar region.

Since admission the child has been treated by iodide of potassium, gr. v, thrice daily, hyd. c̄ creta, gr. j thrice daily, and bromide of potassium. The drug treatment has apparently been of no use whatever. In addition to that he has been treated by frequent nasal syringings of compound alkaline lotion with carbolic acid half a drop to the ounce. These nasal irrigations have been of service to him. The stridor has been very much less when he is awake, and when he is asleep the respiration is quiet. His condition at the time of exhibition is that there is not so much nasal discharge, there is slight improvement in the breathing, and *he sleeps well and quietly.*

(*Exhibited March the 20th*, 1908.)

Discussion.

Dr. Edmund Cautley thought the proper treatment was to remove the adenoids. With regard to the idea of congenital laryngeal stridor, Dr. Carpenter said the stridor did not begin until two weeks after birth, and it was not very severe, nor was there any history of severe attacks of dyspnœa. A gutter-shaped epiglottis was of no value in diagnosis, for in babies it was often of that shape, even in the absence of any laryngeal defect. He thought congenital laryngeal stridor was due to a defect in the formation of the larynx, which disappeared as the larynx increased in size. In the present child the stridor might depend on a catarrhal condition of the naso-pharynx extending downwards in consequence of adenoids. Recently he had in hospital a child, aged 18 months, with a history of hoarseness and dyspnœa of more than a year's duration, dating almost from birth, and with slight stridor. He was convinced it was due to a chronic catarrhal condition. Another baby was so bad with hoarseness and dyspnœa that after consultation it was decided to open the larynx to see if it contained some papillomatous growth. Nothing was found, and two or three years later the child had recovered completely, and eventually had a normal voice.

The Chairman (Dr. C. W. Chapman) said he had a similar case in quite an infant, which was almost suffocated, and he performed tracheotomy. It went on very well, but he was sure there was some growth around the cords. The child was not well looked after, there was some suppuration round the tube, and the patient died. Post-mortem, there was found a papillomatous growth on both sides.

Dr. George Carpenter, in reply, said he did not think it was due to laryngeal catarrh, because the cry was normal. If the voice had been hoarse it would have suggested catarrh of the cords, perhaps an ulcerous condition, syphilitic or tuberculous—and he had met with examples of both these conditions in infants—or a papillomatous growth. There was another possibility, as shown in a case he had many years ago and which he published in the 'Lancet'—the first endo-laryngeal operation he had performed. The girl had a subglottic fibrous tumour, with a long pedicle, and when she breathed or phonated the tumour wedged itself in between the vocal cords. In consequence she was dysphonic. He removed it by means of Morell Mackenzie's forceps, with a satisfactory result. Possibly the present condition might be due to reflex spasm. He was not convinced that there were adenoids, he had found it impossible to make a digital examination with the index finger. Examination of the posterior nares with the little finger afforded him, personally, no assistance or information. The only thing that could be done was to pass a Gottstein's post-nasal curette, and that he would do. Some result might be looked for from that procedure, but he was not hopeful, and did not think there were any adenoids or that the laryngeal stridor was occasioned by these growths. But he would promise Dr. Cautley that he would make the attempt and settle that question.

Postscript.

On March the 27th a note occurs to the effect that the infant's cry remains natural, but there is a great deal of respiratory gurgling with sinking in of the lower interspaces and the episternal and supra-clavicular regions, and much thin muco-pus in the mouth, which can still be seen trickling down the posterior pharyngeal wall. The mouth and throat are normal. He is pale and well-nourished. Nasal syringing brings away large quantities of discharge. The breathing is then greatly increased in frequency and there is much gurgling and spluttering, but no laryngeal spasm. Perhaps on these occasions there is a little hoarseness, but certainly not at other times. Another attempt was made to digitally examine the posterior nares, but it was, as before, found to be impossible of accomplishment.

On April the 1st Gottstein's post-nasal curette was passed into the posterior nares, but the region was free from adenoids. The discharge was as bad as ever. On April the 16th he passed a very bad night and the house-physician was called to him in the early morning. He was very pale, the respirations were feeble, and he had sibilant rhonchi all over the chest associated with falling in of the lower ribs and inter-costal spaces. There was no rise of temperature; on the 13th it had risen to 101·2° F. and on the 14th to 100° F., but for some days prior to that it had ranged between 99·8° F. and 96·4° F. as the lowest. He died the same night and it was impossible to obtain a post-mortem examination. There was a loss of 6 oz. in weight during his stay in hospital.

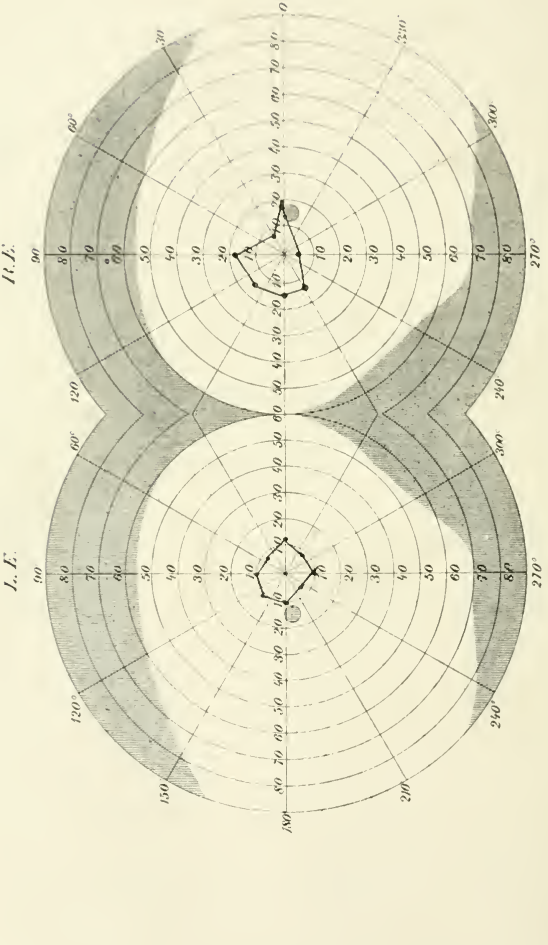
68. A CASE OF JUVENILE TABES DORSALIS.

By SYDNEY STEPHENSON, C.M.

Kate W—, aged 14 years, fell under my notice at the Kensington General Hospital for the first time on February the 26th, 1908.

Personal history.—Nothing known to be wrong with the patient's sight until she had reached the age of ten years,

Fig. 50.



when her school-teacher sent to say that the child was unable to see well. She was sent to school at four years, and during school life, which had terminated shortly before I saw the patient, was a dull child. She reached, however, the fifth school standard. Upon the complaint of bad sight she was taken on October the 14th, 1903, to the Moorfields Hospital, where spectacles were prescribed, and worn for about a year without any particular benefit. On October the 20th, 1904, patient attended the Royal Westminster Ophthalmic Hospital, the out-patient letter of which I have inspected. The sight was then less than $\frac{6}{60}$ with each eye, and a diagnosis of "post-neuritic atrophy" was marked on the sheet. Spectacles (+ 1·0 D. sph.) were prescribed for reading, but again they gave little help.

Present state.—A well-grown but rather dull girl, who has not yet begun to menstruate. $R.V. = < \frac{6}{60}$. $L.V. = < \frac{6}{60}$. The optic discs are pallid and cupped. They present all the appearances of so-called "simple" atrophy —that is to say, they are clearly defined, the markings of the lamina cribrosa can be recognised, the retinal vessels are of good size, and the surrounding retina shows no morbid changes. No choroiditis or perivasculitis; no fine vessels can be found in the corneæ. The visual fields (estimated by means of a 10 mm. white square) are very small, especially in the left eye, where the field does not exceed ten degrees in any direction. The pupils, 5 mm., although sluggish to light, react promptly and well to accommodation—potential Argyll-Robertson phenomenon. The girl shows no ataxy, but easily tires. Bowels regular without recourse to medicine. Imperative desire to urinate. The right knee-jerk cannot be elicited, even on reinforcement (Jendrassik). An occasional jerk can be obtained from the left knee. Ankle-jerks absent. No definite account can be got of "lightning pains."

Family history.—Mother, aged 44 years, in good health. Father, aged 44 years, said to be a strong man in rude health. Two miscarriages. Seven children born alive, of whom the patient, Kate, comes third. Three of the children have died—one at a year from pneumonia, one at two years from phthisis pulmonalis, and the third at three months from bronchitis.

Remarks.—Diagnosis in the foregoing case rests mainly upon: (1) The state of the pupils and of the optic discs; (2) the absence of one knee-jerk and the impairment of the other; and (3) the imperative desire to urinate. It is by no means a typical case, but on any other view than that of juvenile tabes it would be difficult to account for the bilateral optic atrophy, which, clinically at all events, is now certainly not post-neuritic in character, whatever it may once have been.

(*Exhibited March the 20th*, 1908.)

69. A CASE OF HYDRONEPHROSIS.

By GEORGE CARPENTER, M.D.

THE child, a boy, aged 2½ years, was admitted into the Evelina Hospital for Sick Children on January the 25th,

Fig. 51.

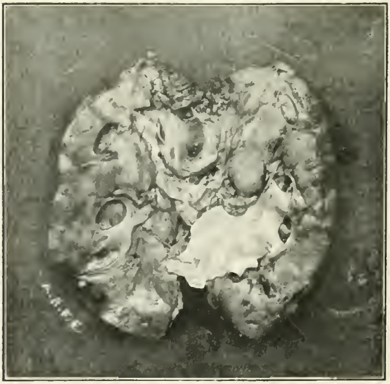


Fig. 52.

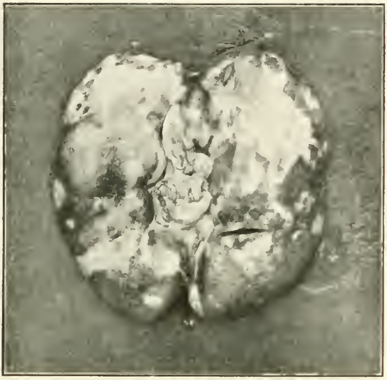
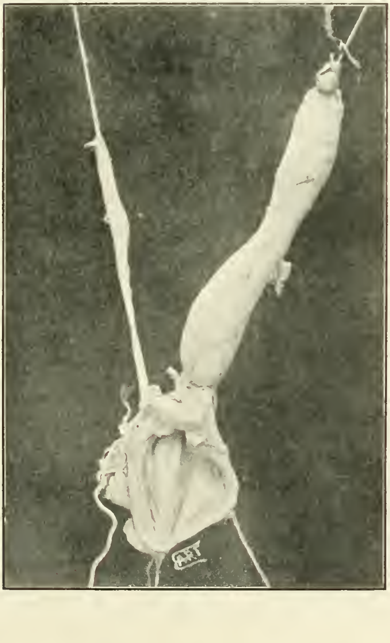


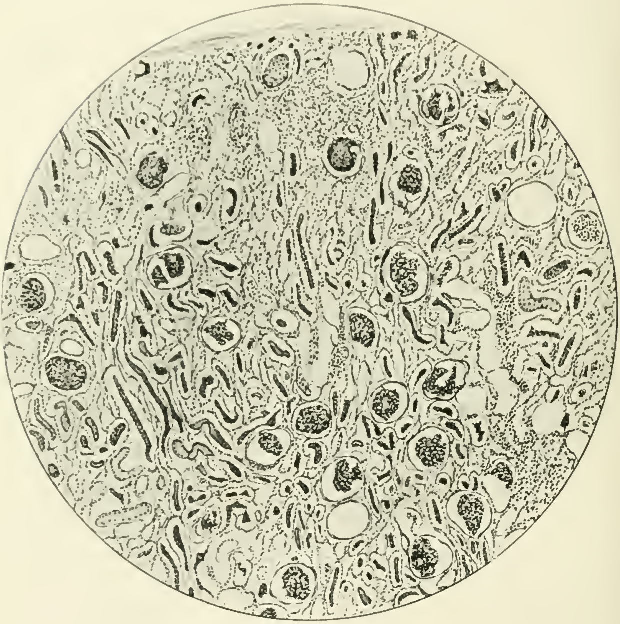
Fig. 53.



1889, with pertussis and double broncho-pneumonia, and died the same day.

The left kidney was mottled all over, pale and violet on its surface and on section (*vide* Figs. 51 and 52).

FIG. 54.



The left ureter was much dilated (*vide* Fig. 53), the constriction being close to its junction with the bladder.

The pelvis and calices were dilated (Fig. 51). The papillæ were natural. The mucous membrane was intact.

Sections of the kidney showed patches of round-celled infiltrations and unevenly distributed fibrosis, with compression and destruction of the renal parenchyma (Fig. 54).

There were signs of dilatation in some of the cortical convoluted tubules.

(*Exhibited March the 20th,* 1908.)

70. A CASE OF CEREBRAL DIPLEGIA.

By GEORGE CARPENTER, M.D.

Thomas H—, aged 6 years, the third child, was born at full term. The confinement was straightforward; the head presented, but help was not obtained until after his birth. He appeared to be healthy until eight months of age "when he walked a few steps and then straight away went back."

He now cannot walk, and can only say "mam" or "dad," and is not clean in his habits. He had measles, whooping-cough, and chickenpox some eighteen months previous to his admission into hospital on February the 17th, 1908.

Four other children, all healthy. No miscarriages.

On examination he is a stupid-looking boy, with oscillating nystagmus associated with jerking movements of the head, which become marked if he tries to fix any near object. His perception for near objects appears to be all right.

The grip of both hands is weak, the right more than the left, but he can move his arms quite freely. The hands, which are cold and blue, are inclined to drop at the wrist, but he can move and use them. When stretching out his hands he keeps the little and the ring fingers flexed. They become tremulous under forced effort, *e. g.* squeezing very hard. The supinator and triceps jerks are present on both sides, but not very active.

He cannot stand. His feet are crossed over one another, the toes pointing to the opposite side from adductor spasm, and only his toes and part of the outer edge of the feet touch the ground. Talipes equino-varus is especially marked on the right side. The knees are flexed; the knee-jerks are exaggerated. Babinski's sign is present. Ankle clonus not obtained. The feet are blue and cold.

The optic discs are not well defined, but there are no coarse changes in the fundi oculorum.

(*Exhibited March the 20th*, 1908.)

71. INFANTILE HÆMIPLEGIA WITH ATHETOSIS OF ARM.

By F. W. HIGGS, M.B.,

AND

P. TURNER, M.S.

A. S—, female, aged 8 years and 9 months, first seen on March the 9th, 1908.

History.—First child normal labour. No illness until the age of ten months, when the child had a "slight fit," and during the following fortnight fourteen severe fits occurred. The mother states that during the fits the child was unconscious, twitched, and had the mouth drawn over to the right side. At about the same time as the fits occurred the child is said to have had whooping-cough, but this is very doubtful (the child apparently never whooped). Three months after the fits occurred the mother observed that the right arm "seemed stiff." No attempt at walking was made before eighteen months, and the child could not walk by herself until two years of age, and then was observed to walk lame. It was not until this time that she commenced to talk.

Since the first attack of fits the patient has had numerous fits at varying intervals, the longest interval being four years, from three to seven years of age. Latterly the fits have been less severe. The child now knows when fits are coming on, as they always commence with twitching of right side of mouth. Recently this twitching has occurred sometimes without subsequent unconsciousness.

Present condition.—Patient is a well-nourished girl of quite average intelligence. There is an old, partially recovered left hemiplegia; paresis of lower part of face, some paresis of leg sufficient to produce dragging of the limb and cause the child to walk slightly lame. The foot "gives way" and turns inwards when she attempts to walk quickly. There is slight talipes equino-varus in that foot. The arm exhibits spastic paralysis with some rigidity and well-marked athetosis of the hand and forearm.

The tendon reflexes are brisk.

It was at first thought that the hemiplegia might have been due to a cerebral hæmorrhage occurring in the course of whooping-cough, but the history and course strongly support the view that the original lesion was one of acute encephalitis.

(*Exhibited March the 20th*, 1908.)

72. THE ALL-FOURS EXERCISER IN THE AFTER-TREATMENT OF EMPYEMA IN A BOY AGED THREE YEARS.*

By WILLIAM EWART, M.D.

A BRIEF statement of the clinical history will suffice, as this does not present any unusual features except the use of the method which has to be demonstrated. The clinical dates in the case are as follows:

December the 16th, 1907: Sudden onset of illness with a slight convulsion in a little boy aged 3 years. The patient was ill and confined to bed with bronchitis and pneumonia at home until admission. January the 22nd, 1908: Admitted into the Belgrave Hospital with right pneumococcus empyema. January the 23rd: Aspiration of pus. January the 24th: Operation by Mr. Philip Turner, who resected one inch of the ninth rib in the posterior axillary line. March the 6th: Cutaneous wound healed. March the 7th: Got up for the first time. March the 9th: The following condition was found on examination:

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* Demonstrated before The Society for the Study of Disease in Children, on March the 20th, 1908.

General configuration of the chest broad and flat, with striking convex prominence of the cardiac area to the left and also slightly to the right of the sternum. This is the only portion of the right front participating in the active respiratory movements of *the left chest,* which shows considerable excursions of the left costal arch and of the left shoulder. But the right clavicle and trapezius are at a lower level than the left; the supra- and infra-clavicular fossæ are depressed and motionless, and the right mammary region is also flat and motionless. The infra-axillary surface is angular, instead of normally convex like the left.

Posteriorly the disparity in size is slighter, but the right chest moves much less than the left. The abdomen is toad-like in shape.

Auscultation.—Anteriorly: Left, good; right upper, expiration tubular. The lower half, inspiration and expiration blowing. Heart-sounds normal, but the position of heart as ascertained by X ray is still "median." *Posteriorly:* left, active puerile breathing; right, in upper half, respiration almost tubular and in the lower part weak.

The exerciser was first used on the 10th, and it was since then used daily for gradually increasing periods of time. On the 14th considerable improvement was already perceptible in the aspect and movement of the right chest. 20th: At the present date the right chest is still imperfectly expanded, but great progress has been made since the examination on March the 9th, both as regards the size of the right chest and the amplitude of its movement.

The all-fours exerciser and its smaller pattern, the *infant-rest or baby-rest,* were first shown before The Society for the Study of Disease in Children on May the 3rd, 1907.* I have since then adapted the exerciser for the after-treatment of empyema in children, by providing the trough (in which the patient is safely belted in the prone position as though on a swimming board) with a longitudinal axle which allows the trough with the child in it to be turned and fixed into any degree of lateral posture. The desired complete lateral decubitus on the sound side is thus obtained, and meanwhile the child has the use of his four limbs, much to the advantage of his re-expanding the lung. This *complete* lateralisation is a distinct improvement upon the analogous opportunities offered for adults by the "convalescent machine," the larger pattern of the exerciser, which was first exhibited before the Polyclinic on October the 20th, 1907.†

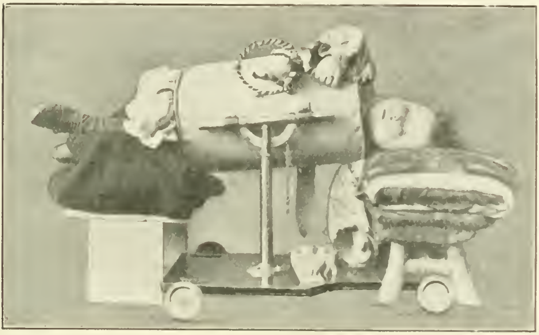

FIG. 55.—Showing metal trough, lined with canvas, and mechanisms for varying posture. (Supports improvised in the studio.)

The use of the convalescent machine and of the all-fours exerciser in bronchiectasis was set forth on March the 17th, 1908, before the East Sussex Medical and Chirurgical Society in a paper on the "Principles of Treatment of Bronchiectasis." For this condition "inclination" is the requirement, not lateralisation; and this is obtained in the convalescent machine by raising the level of the posterior part of the trough or body-support above its anterior level, so that in the prone posture the patient's shoulders slant downwards. In the all-fours exerciser the same facility is afforded by a transverse horizontal regulating gear.

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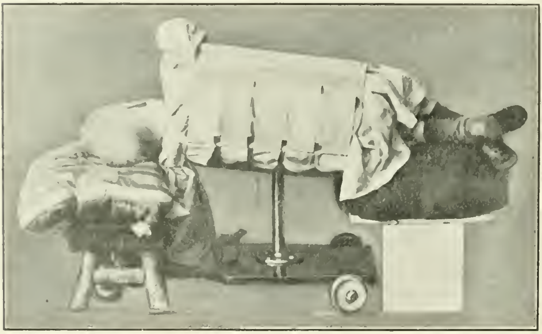
* 'Reports,' vol. vii, p. 201.

† *Cf.* Medical Press and Circular,' February the 19th, 1908.

FIG. 56.



For empyema the special advantage of the exerciser is that it enables us to begin the treatment for re-expansion on the second or third day, or as soon as "surgically" possible after the operation: (1) At first the child is simply rested in it, with suitable supports for the head and legs; and (2) a little later allowed graduated exercise. All that is then needed is to remove the supports and to set the limbs and the head free for given periods, with appropriate intervals of rest. This *re-expansion treatment* was delayed in this instance by surgical complications. I regard this as an improvement upon the two methods which I devised and practised some years ago—the "empyema mattress treatment" and the "hammock treatment."*

(*Exhibited March the 20th,* 1908.)

73. HENOCH'S PURPURA AND INTUSSUSCEPTION.

By HUGH LETT, Ch.B.

James P—, aged 3 years, was admitted to Poplar Hospital on Sunday, September the 29th, 1907. The following history was obtained from his mother.

He was quite well until Friday, September the 27th, and had been playing about as usual until 4 p.m., when his mother gave him a bath. He then complained of pain in the left leg, and his mother noticed that the knee was swollen. The same night the left elbow was swollen. The next morning there was considerable swelling of the scrotum and both legs.

No spots were seen on Saturday afternoon, September the 28th, but in the evening the mother noticed one or two "purple watery blisters" on the left leg. The left ankle was then so swollen that his sock had to be cut before it could be taken off.

The bowels were opened naturally until Friday the 27th. On that day they were opened frequently, the motions being only partly formed. On Saturday morning he had one natural motion.

He did not complain of abdominal pain until Saturday morning, when he was suddenly seized with a paroxysm of pain which made him scream. Similar attacks of pain associated with screaming fits recurred at intervals until his admission to hospital on the following day. Blood and mucus were passed *per anum* on Saturday evening and this was repeated several times during the night. He vomited once on Saturday morning after screaming with pain.

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* "Sketch of an Ideal Medical Treatment of Empyema," 'The Polyclinic,' vol. iii, 1900, p. 113.

There was no history of sore throat; he had never had scarlet fever or any other illness, except measles when he was six months old. There was one other child at home at the time who was quite well.

On admission the temperature was 99·4° F. and the pulse-rate 140 per minute. The boy looked ill, and there were small purpuric patches on the ears, legs and scrotum.

The abdomen was slightly distended, though not tender, and palpation revealed a sausage-shaped swelling in the right lumbar region. The spleen could not be palpated.

On making a rectal examination nothing abnormal was felt, but when the examining finger was withdrawn it was covered with blood.

A small motion passed after the child's admission to the Hospital was examined and found to consist of blood and mucus only, without a trace of bile.

Laparotomy was at once performed. A two-inch incision was made through the right rectus muscle, and an ileo-cæcal intussusception found. This was reduced without difficulty, and the wound closed with through-and-through sutures of silkworm gut.

Subsequent history.—On September the 30th, the day after the operation, the boy appeared to be much better. He had had a normal motion, the vomiting had ceased and there was no abdominal pain.

The purpura, however, was a little more marked. The patches were of irregular contour though small in size, the majority being not more than half an inch in diameter, but they were beginning to become inflamed and to slough. They were then scattered over the face, ears, scrotum and legs.

On October the 2nd diarrhœa set in, and during the twenty-four hours there were nine motions. There was no vomiting, but the stools contained blood. There was some abdominal pain.

By October the 3rd the purpura had considerably increased; there were large patches on the face, hands, and ears, which were turning black and beginning to form blebs. Small spots had appeared on the legs, thighs, and forearms, but there were none on the chest or abdomen. The diarrhœa had ceased and the stools were free from blood, though they were still loose.

On October the 6th the boy vomited four times and complained of abdominal pain again.

On October the 7th the purpura was still more pronounced on the face, hands, and legs. The right hand was swollen out of recognition. It was black and the fingers were becoming gangrenous. The left hand was swollen and œdematous between the purpuric patches.

The abdomen was distended, the laparotomy wound was purpuric and sloughing, and a coil of intestine was seen lying at the bottom of the wound. Vomiting was persistent, and the stools were composed of blood and mucus.

The abdomen became more and more distended, and the presence of free fluid in the peritoneal cavity was demonstrated.

The patient died on October the 9th.

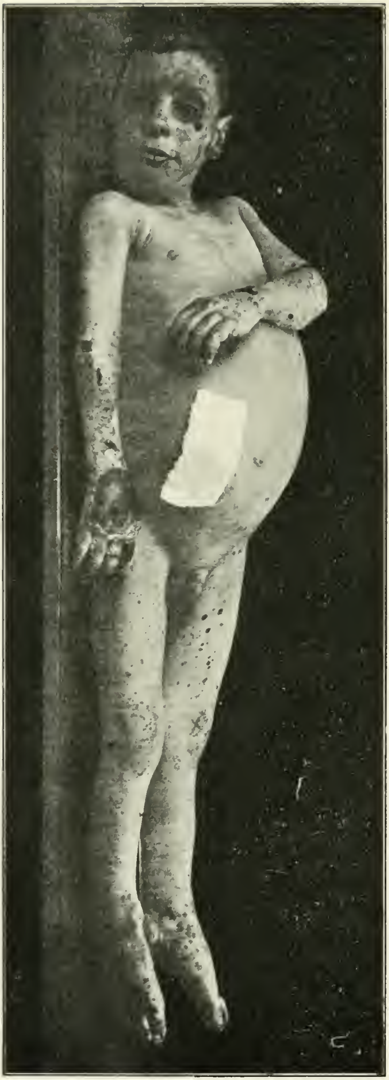
At no time did the vomit contain blood, the urine was free from blood and albumin, and the spleen was not palpable. The temperature varied between 99° F. and 102·4° F. from October the 4th to October the 7th, and then remained at about 99° F. until the end.

Post-mortem.—There was general septic peritonitis. An enteric intussusception was found eighteen inches from the ileo-cæcal valve. It was five inches long and irreducible.

The spleen and other solid viscera were normal, and no submucous hæmorrhages were found in the intestines. Peyer's patches were not enlarged. On laying open the intussusception the intussusceptum was seen to be intensely congested and thickened towards its apex, but it was not possible to say that the intussusception had been directly caused by a sub-mucous hæmorrhage.

The above case, briefly epitomised, is as follows: Frequent stools, partly formed, on September the 27th; the same evening pain and swelling about the left knee and

FIG. 57.



later about the left elbow. The next morning swelling of the scrotum and both legs, and severe paroxysms of abdominal pain, probably due to the formation of the intussusception. In the afternoon what are described as one or two purple watery blisters were seen on the swollen left leg. The following day the intussusception was operated upon and reduced. The purpura increased; there was passage of blood *per anum* three days later; four days later a second intussusception occurred. Death three days later.

The case was one of Henoch's purpura, complicated by the presence of two separate intussusceptions occurring at an interval of nine days.

I do not propose in the short time at my disposal to enter into an elaborate consideration of Henoch's purpura: suffice it to say that Henoch (1) in 1874 brought forward a number of cases of purpura which were associated with intestinal colic, vomiting, intestinal hæmorrhage, and frequently painful swelling of the joints. Important articles have since been written by v. Dusch and Hoche (2) Sutherland (3) and others, showing that Henoch's purpura is a definite clinical entity. Other communications on the subject have been made by George Carpenter (4), Couty (5), Vierhuff (6), Osler (7), and Bristowe (8), while cases have been recorded by Burrows (9), Silberman (10), Raymond Crawfurd (11), Theodore Fisher (12), Zimmerman (13), and others.

The object of this paper is to consider Henoch's purpura from a surgical point of view and to discuss chiefly the question of diagnosis. Firstly, the differential diagnosis between Henoch's purpura and intussusception, and secondly, the diagnosis of an intussusception occurring during an attack of Henoch's purpura. That there is room for such consideration is proved by the reporting, not only of cases of Henoch's purpura which were operated upon as cases of intussusception when there was no intussusception, but also of cases in which an intussusception occurred during an attack of Henoch's purpura but was not recognised until the patient arrived in the post-mortem room.

At first sight it would appear that the diagnosis of Henoch's purpura presents no great difficulties, and that to anyone acquainted with the disease it should be easy to distinguish between it and intussusception. Unfortunately this is by no means the case, for as in Dr. Sutherland's (3) first case, the abdominal pain and vomiting may occur some time before the appearance of any cutaneous lesions. Theodore Fisher (14) has described a case of Henoch's purpura which proved fatal, and in which there were no hæmorrhages into the skin. Osler (15) has also pointed out in an article on the visceral lesions of the erythema group that visceral crises may occur independently of the skin lesions. A purpuric eruption, however, joint pains, hæmaturia, vomiting of blood, or a history of a previous similar attack, should remind one that the patient may be suffering from Henoch's purpura. In the absence of all these signs it may also be remembered that whereas Henoch's purpura is rarely met with in children under three years of age, and generally occurs in children several years older than this, the great majority of cases of intussusception occur in babies. Out of 187 cases of intussusception Mr. Barnard (16) found that 135, or 72 per cent., were under one year old, and Mr. McAdam Eccles (17) obtained a very similar result from a smaller number, viz. out of 40 cases, 27, that is, 68 per cent., were under one year. Mr. Fitzwilliams (18), again, in 648 cases under the age of twelve years, found that 466, or 71·9 per cent., occurred in children not more than twelve months old, and only 6 per cent. between the ages of seven and twelve years.

The joint pains are, as a rule, temporary, and pale into insignificance when the abdominal symptoms arise. They are, however, present in a considerable proportion of cases, v. Dusch and Hoche finding them in 40 out of 44 cases recorded by them.

The character of the onset of the abdominal pain will not materially help the diagnosis, as in both intussusception and Henoch's purpura it is sudden and may be preceded by diarrhœa. I have, however, operated upon two cases of intussusception which were immediately preceded by, and apparently caused by, a fall on to the abdomen.

In both conditions the pain is paroxysmal and extremely severe; between the paroxysms the patient is fairly comfortable, though in the later stages of intussusception the pain becomes more continuous. A slight rise of temperature is not uncommon in Henoch's purpura, but may also be met with in intussusception.

Vomiting is not an urgent sign in intussusception until the later stages, and though the child may vomit once or twice at the onset, it usually ceases or is only very occasional until the condition becomes very grave. If severe vomiting is present from the beginning, and especially if the vomit contains blood, the probabilities are against the case being one of intussusception. Mr. Harold Burrows (9) is strongly of opinion that his patient had fæcal vomiting before admission to hospital, though there was none after admission, and he alludes to another case in which fæcal vomiting is said to have occurred, but we must wait for further evidence on this point before we can draw any conclusions from these two cases. Tenesmus is an early symptom in intussusception, and frequent tenesmus with the passage of mucus and blood *per anum* is in favour of intussusception, though tenesmus may occur in Henoch's purpura. Mr. Barnard (19) has pointed out a sign which is of considerable value in the diagnosis of acute intussusception in babies; the blood and mucus on the napkin are carefully examined by the naked eye for the presence of bile, and if bile is absent he considers it a proof of the presence of an intussusception. In thirty-seven cases of intussusception at the London Hospital bile was only present on the napkin on one occasion; the sign therefore only failed once. In four cases of acute ileocolitis which were admitted to the hospital as possibly cases of intussusception, bile was seen on every occasion.

Unfortunately this test does not hold good in older children.

But it is in the local abdominal signs that most interest centres. In Henoch's purpura as a rule the abdomen is not tender, but in various cases it has been described as being "distended and resistant" (Sutherland) (3), "extremely tender on pressure" (Theodore Fisher) (12), and as being not distended but "generally tender and rigid, especially in the lower part" (H. Burrows) (9). In intussusception the abdomen is neither distended nor tender at first, though the intussusception itself is tender, and manipulation will cause it to harden and will bring on an attack of pain.

There is yet one more sign to which I wish to allude. It is, I believe, the crucial test, and the one which should be relied upon above all others in making a positive diagnosis of intussusception. I refer to the presence of a tumour. Statements vary as to the frequency with which a tumour can be felt. Rose and Carless (20) say that a tumour can be felt in about half the cases. In Mr. McAdam Eccles' (17) forty cases, a tumour was found in thirty-two, that is, in 80 per cent. Mr. Barnard (21) says that an abdominal tumour will be found in 75 per cent. of the cases if an anæsthetic is given; and Mr. H. S. Clogg (22) found a tumour in fifteen out of sixteen cases. It is possible, however, that if a careful examination is made by the rectum and abdomen, with an anæsthetic if necessary, a tumour will be found still more frequently. I have operated upon twenty-four cases of intussusception, and in every case I have been able to feel a tumour before operation, either by the rectum or through the abdominal wall. In a few cases, however, it was necessary to anæsthetise the patient before the tumour could be found. The tumour is sausage- or kidney-shaped, and during an attack of pain becomes more definite. It is freely movable and can often be partially reduced by manipulation through the abdominal wall, slipping from the grasp in a very characteristic way.

I know of one case in which hæmorrhage into the mesentery was thought to be an intussusception, and have heard of another in which a collection of coagulated blood within the lumen of the gut was similarly misinterpreted. Hæmorrhage into the intestinal wall may produce a tumour which can be palpated, but in most cases it will lack certain features which are characteristic of an intussusception. For instance, it will not have the slight curve which is produced by the drag of the mesentery on the intussusceptum, it will not contract and become more obvious on palpation, nor will it be possible to partially reduce it by manipulation through the abdominal wall.

A very interesting case has been recorded by Greig (23). The patient was a boy, aged 9 years. Six days before his admission to hospital he vomited, and the following day complained of pains in the legs and had some diarrhœa. Two days later he was better, but the left knee was swollen. The following day the abdominal pain and diarrhœa returned and a few purpuric spots were seen on the legs. Two days later he was admitted to the hospital. He was then vomiting small quantities of brownish-yellow offensive fluid. The abdomen was tense and rigid, but not distended, the rigidity being most marked below and to the right of the umbilicus. Under chloroform, a small firm tumour was felt in the right iliac fossa. It was freely movable and slipped away from between the fingers. No purpuric spots were observed then. Laparotomy was performed, and the tumour was found to be due to great congestion of the lower end of the ileum, which projected like a collar into the cæcum. The abdomen was closed and the wound healed by first intention. Two days after the operation the left side of the face was much swollen and the left eye almost closed. Later, this became discoloured and evidently purpuric. Several days later there was a profuse purpuric eruption on the hands, legs, and body.

This case shows how extremely difficult it may be to distinguish between hæmorrhage into a limited portion of the intestine and a very short intussusception. In this case it was only actual inspection and palpation of the gut that determined the diagnosis.

The tumour, of course, was a small one, but there can be no doubt that the length of an intussusception may vary greatly from time to time, the intussusceptum not only advancing but also receding to a considerable extent, and possibly spontaneous reduction may take place. This, at any rate, is the explanation usually accepted when the surgeon, after making a diagnosis of intussusception, opens the abdomen and finds no tumour but only congestion of the lower end of the ileum and adjoining part of the cæcum. But, as Dr. Sutherland asked in the discussion which followed the reading of this paper before The Society for the Study of Disease in Children, may not some of these cases really be examples of Henoch's purpura in which the cutaneous lesions are absent?

So much for the differential diagnosis between Henoch's purpura and intussusception; but it may be necessary to diagnose an intussusception which has occurred during an attack of Henoch's purpura. Dr. Sutherland (3), in a valuable paper in 'Pediatrics,' has recorded an example of this, and Vierhuff (6) has recorded a case of purpura hæmorrhagica in an adult, who developed an intussusception which was ultimately passed *per anum*. Here, again, diagnosis must rest on the presence or absence of a tumour. In the case of James P—, which I have described above, there was a definite sausage-shaped tumour, and although the purpura was noticed and the possibility of the hæmorrhage from the bowel being due to Henoch's purpura fully recognised, an intussusception was diagnosed from the presence and character of the tumour. As Sutherland suggests, severe tenesmus, if present, is also a sign of considerable value.

One other point of interest in my case may be alluded to: the wound showed no signs of healing but became purpuric and ultimately gave way entirely. I have also heard of another case where the child died from hæmorrhage from the wound. On the other hand it must be admitted that the wound has in other cases healed well by first intention.

The occurrence of a second intussusception a few days after the reduction of the first is most unusual. That the intussusception found on post-mortem examination was not merely a recurrence of the original intussusception is proved by the fact that the first one was ileo-cæcal, whereas the second was enteric, and situated ten inches from the ileo-cæcal valve; also by the facts that the vomiting and pain ceased immediately after the operation, that the bowels were opened, and that the child was free from all signs of intussusception for seven days. Unfortunately, when the second intussusception occurred the child's condition was such that any further operation was out of the question.

In conclusion, there are a certain number of cases in which the diagnosis is extremely difficult. The greatest difficulty is perhaps presented by those cases in which a diagnosis of Henoch's purpura has been made, but it is doubtful whether an intussusception is present or not. The wisest plan to adopt is not to operate unless a definite tumour can be felt. For many of the symptoms of an intussusception, viz. vomiting, tenesmus, blood and mucus in the stools may be present, although there is no intussusception. If, when the diagnosis is made, the skin lesions are extensive, reduction should be attempted by means of injection, and only when this has failed should laparotomy be performed as a last resort.

It may be suggested that an exploratory laparotomy is a small matter, and should be performed if there is any doubt as to the diagnosis, but the subsequent condition of the wound may be so serious that a laparotomy is not to be lightly undertaken. Intussusception is a rare complication of Henoch's purpura, and further, in the great majority of cases of intussusception a tumour can be palpated; when, therefore, the risks associated with a laparotomy wound are also taken into consideration, it seems obvious that if symptoms of intussusception make their appearance during an attack of Henoch's purpura operation is inadvisable, unless a characteristic tumour can be palpated.

Apologies are perhaps due from me for the length of this paper, but I think that the matter is one of some considerable importance, and so little has been written on the subject that I feel justified in going into the matter in some detail before this Society, which has done so much to encourage the study of disease in children.

I must express my indebtedness to Mr. H. E. Ridewood, late Senior House-Surgeon at the Poplar Hospital, for many of the notes of the case, and also to Mr. Hugh M. Rigby, for whom I was acting when the case was admitted to hospital, for permission to publish it.

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(*Read April the* 10*th*, 1908.)

Discussion.

The Chairman (Dr. Edmund Hobhouse) (Brighton) said the case which Mr. Lett had narrated was of great interest, and the author had dealt very fully with all the questions which arose in connection with it. An interesting point was whether the intussusception was caused by hæmorrhage into the mucous membrane of the intestine. Apparently there were no such hæmorrhages discovered post-mortem, so that did not seem to have been the case. The whole question of the relation of intussusception to Henoch's purpura was very interesting. Last summer he had a case in a child, aged 3 years, in which it was practically impossible to make up one's mind. The child was left for twelve hours, and the symptoms cleared up. There had been preliminary diarrhœa before other symptoms appeared; it was not possible to feel a tumour, and the parents would not consent to an anæsthetic being given. He agreed with Mr. Lett that in doubtful cases it was desirable to make an exploratory incision, but in his, Mr. Lett's case, he himself pointed out a condition which would make one shy of such a course, namely, the strong purpuric tendency. The only cases in which the diagnosis was evident were those in which there was a skin lesion. Intestestinal hæmorrhage often occurred long before purpura on the skin.

Dr. G. A. Sutherland agreed as to the interest of Mr. Lett's paper, and thought it was the first occasion on which this subject had been brought before the Society. Many diagnostic signs had been given by the author, but as he, Dr. Sutherland, had been quite wrong in several cases, he felt that however easy it might be to lay down diagnostic signs, it was a difficult matter to make an accurate diagnosis at the bedside. His experience had led him to certain conclusions. First, in the case of purpura with hæmorrhage into the intestinal wall, all the symptoms of intussusception might be produced. In many cases no tumour was found. He maintained that all the signs of intussusception might be produced without that condition being present. As Mr. Lett's case showed, intussusception might occur in the course of Henoch's purpura, and probably hæmorrhage into the bowel wall was directly a cause of intussusception. Such hæmorrhage was often localised in the ileo-cæcal region, producing a paralysis of the bowel wall there, and probably as a result of that the intussusception took place. Many cases were recorded in the literature, but there had not been a satisfactory explanation of the ætiology. From his experience he would hesitate very much to operate if there were other signs of purpura present in connection with the symptoms of intussusception, because it had been shown that all the symptoms might be associated with the hæmorrhage into the bowel wall pure and simple. In two cases he had had the bowel investigated for intussusception and none had been found. He believed there was a site of election for hæmorrhage into the bowel wall, namely, the lower end of the ileum, approaching the ileocæcal valve. In one case last year he had the abdomen opened, but no intussusception was found, but there was extensive hæmorrhage into the last two inches of the ileum, and into the cæcum and first part of the colon. The child died thirty-six hours afterwards, never having rallied from the acute attack, and there had been persistent vomiting. There had been extensive hæmorrhages into the submucous tissues of the bowel wall, and there were scattered petechiæ. All had probably diagnosed cases as intussusception, in which, when laparotomy was done, the condition was found to have undergone spontaneous reduction. He used to accept that view but was now very sceptical about spontaneous reduction. He doubted whether such an extensive intussusception as some of the cases must have been could have undergone spontaneous reduction, and thought that some at least of these cases were examples of intestinal hæmorrhage only. Operation was not without danger in such cases, because the hæmorrhagic diathesis might be present, and bleeding might persist from the operation wound. In one case a hot fomentation was put on, and next day the abdominal wall was black from hæmorrhage. The pathology of Henoch's purpura was obscure, but he thought it probably was an infective disease; that first there was some invasion of the bowel wall, the results showing themselves locally at first, and then general symptoms supervened, such general symptoms being shown by the frequent effusions into joints, purpura, erythemata, etc., and if the affection persisted, by hæmorrhagic nephritis.

Mr. Duncan FitzWilliams said Mr. Lett's case was such an unusual one that it was suitable for discussion. Most people recognised that the symptoms of Henoch's purpura might simulate those of ordinary intussusception, and the presence of a tumour put the matter beyond reasonable doubt. He agreed that if an anæsthetic was given and a careful examination was made, a tumour would be found in 85 to 90 per cent. of the cases. But he had seen cases in which a tumour was present, but the most careful palpation failed to discover it; it might be under the liver. At Great Ormond Street he had seen a case of Henoch's purpura, which came in with all the physical signs of intussusception. A definite tumour was felt in the right iliac fossa. Operation was performed, but no intussusception was found, and it was thought to have been spontaneously reduced. Large hæmorrhages had occurred in a portion of the lower ileum and adjacent mesentery, and the tumour which had been felt proved to be a large blood-clot in the cæcum. The wound was sewn up, but the child died from oozing from the wound which nothing could stop. He had been present at three other operations in which no tumour was found, and in which spontaneous reduction had apparently been brought about. He had accepted that as the most likely explanation, but was not so sure of it now. Still, he had no doubt that spontaneous reduction did occur. He had seen a case in which the intussusception projected beyond the anus, and by the time the abdomen was opened for no apparent reason it had become reduced to 1½ inches in length. If there was any reasonable doubt he would not operate; he thought that unless there were strong grounds for diagnosing intussusception in place of Henoch's purpura no operation should be done. Wounds did not behave kindly where there was a marked hæmorrhagic diathesis.

Mr. Hugh Lett, in reply, said that there was no doubt that hæmorrhage into the bowel wall could cause intussusception, provided that the projection into the lumen of the bowel was sufficiently great to be caught by the gut below. He repeated that the presence of a tumour was the most important sign there was of intussusception. If there was any reasonable doubt whether intussusception was present he would not operate, but if there were symptoms of intussusception in Henoch's purpura, and there was an abdominal tumour closely resembling intussusception, he would explore. It was a risk, because some cases might bleed and others might slough, but that was not always so. And whereas many cases of purpura got well, the mortality of intussusception when left alone was very great.

74. A CASE OF TETANUS NEONATORUM, WITH SOME REMARKS AS TO CAUSE.

By J. PORTER PARKINSON, M.D.

Walter O—, aged 2 weeks, was admitted on February the 6th into the wards of the Queen's Hospital for "lock-jaw." The mother could not be interviewed, and the father was unable to give any account of the illness except that it had existed for four days. The mother had been pregnant twelve times; there were two miscarriages, and three children died in infancy, two of "infantile consumption," aged 6 days and 3 weeks; another died at six days old from "lockjaw," and was said to have the same symptoms as this infant, being unable to take the breast.

On admission the infant was rigid, so much so indeed that the body hardly bent at all when the hand was placed under one of the heels, and the limb and trunk raised from the bed so that the child rested on its head; there was marked cervical opisthotonos and some of the trunk, the arms were rigid and flexed, the hand clenched with the thumbs inside the fingers, the legs were also rigid and extended, the feet extended and the toes flexed into the sole, the great toe being somewhat widely separated from the others. The rigidity of the face was extreme, there being a well-marked "risus sardonicus," the mouth was tightly shut but widened laterally, the forehead was wrinkled by contraction of its muscles, and the eyes were only partially open. The umbilical wound was not healed and unhealthy looking, discharging a thin sticky fluid. The temperature varied between 99° and 100° F., but later it rose occasionally to 100° and 101° F. The spasm was continuous and did not vary much in degree. Feeding was difficult, though when the teat of a feeding bottle was once inserted between the lips the infant sucked readily, and so a fairly good quantity of nourishment was taken.

During the stay in hospital up to the time of death, on February the 23rd, the rigidity lessened slightly, and it was hoped that the child would recover, but without any warning the infant died suddenly from asphyxia. There had been occasional vomiting during the week preceding death, and the temperature was more elevated during this period.

At the necropsy only signs of asphyxia were found.

It was discovered that the mother had treated the umbilical wound with Fuller's earth, and this was no doubt the cause of infection both of this child and of the previous infant of her family which had died from the same cause. It was to draw attention to the prevalence of this practice that I have brought the case before the

Society, as I find that the custom of using Fuller's earth for the umbilical wound is a common practice, and that this source of tetanus is well-known to coroners. I think, however, that the knowledge should be more widespread.

(*Read April the 10th,* 1908.)

75. TWO CASES OF TETANUS NEONATORUM.

By GEORGE CARPENTER, M.D.

Case 1.—Baby R—, aged 1 week, son of an upholsterer, was admitted into the Queen's Hospital for Children on November the 3rd, 1907. It was breast-fed and took well up to five days old (two days previous to admission) and the "cord separated quite well." Fuller's earth was applied. On the sixth day the mother noticed that the mouth could not be opened. "When it cried it went stiff and threw its head back." When it was admitted it was very rigid, the jaws were fixed continuously, the hands were clenched, but with the feet nothing special was observed, and it did not arch its back. "It had great difficulty in breathing, as if it were going to choke, it got blue in the face, and when it was fed nasally it became black in the face."

On examination there was a muco-purulent discharge from the right nostril and a slight discharge from the navel, which was not inflamed. The latter was examined bacteriologically and the pathologist found in the smears Diplococcus proteus, staphylococcus, but no specific organisms. The fontanelles did not bulge, but the neck was rigid and very slightly retracted. The arms were flexed, the forearms flexed on the arms, and the hands were clenched. The legs were rigid, but the toes were not involved. He was cyanosed and had inspiratory retraction. Some moist sounds were audible over the chest, being possibly conducted sounds from the nose. Both sides of the chest behind gave quite a boxy note to percussion and the laryngeal sounds were particularly well conducted over the left side. When the chest was being examined he had much hiccough and became cyanosed. The mouth was partially open but the jaw was stiff, and when manipulation was made the child became blue, the mouth foamed and the jaw became very decidedly fixed. On these occasions the contracting masseter muscles could be felt to be strongly acting. The day previous the sister in charge of the ward said the jaw was tightly fixed all day and nothing could be got between the lips, the spasm never relaxing, whereas to-day there is decidedly less spasm, and an attempt has been made to feed him by the mouth, but unsuccessfully, as spasm is at once induced by the operation. The child while lying still was fairly comfortable, but any disturbance caused tonic spasm of the face muscles, arms, hands, legs, and trunk; the masseters were markedly affected. During these spasms the infant became extremely cyanosed and the pulse was slow and irregular. Temperature on admission 97·8° F., on the morning of the 4th 97° F., and in the evening 97·6° F., the pulse at this time being 140 to the minute and the respirations 80 to the minute. He was given bromide and chloral āā gr. iij *quartis horis*.

On the evening of the 4th the spasms were more frequent and he was ordered 20 c.c. of anti-tetanic serum, which was injected into the subcutaneous tissue of the abdomen at 6 p.m.

He died at 7 a.m. the following day, the morning temperature being 101·4° F.

At the post-mortem examination there was found collapse of the lungs; the heart engorged on the right side; the brain congested; the tissues round the umbilicus thickened and unhealthy-looking. The other organs were normal.

Case 2.—Baby Brown, aged 1 week, son of an upholsterer, was admitted into the Queen's Hospital for Children on April the 2nd, 1908. Nothing amiss was noticed with the child until tea-time the previous day.

The mother had been attended by a Salvation Army nurse. The cord came away two days after birth; nothing was done to it, there was no discharge from it, and it was dressed with Fuller's earth. The child began to cry and it was thought to be suffering from stomach-ache. Its mouth became stiff, but prior to that it was kicking and clenching its hands. Its mouth subsequently became less stiff, and they got some milk down with a spoon, but later on in the evening it could not take the breast, and the following morning it was admitted at 6.30 a.m.

On examination.—The face is screwed up and risus sardonicus is a prominent feature. The finger can just be inserted into the mouth, which is kept tightly clenched. At times the infant is quite limp and at others the body and limbs become rigid. He is more rigid in the lower extremities than in the upper. When the rigid state passes away there is some twitching. When a spasm comes on he froths at the mouth and gets blue in the face. He is unable to swallow and turns black in the face if milk is put into the mouth. The eyes are kept closed; knee-jerks not obtained; plantar reflex present, no definite Babinski. Heart, nothing abnormal detected; respirations irregular and jerky.

At 5.30 p.m. he was lumbar punctured—no fluid obtained. He was injected with 5 c.c. of anti-tetanic serum (Lister Institute) through the cannula and with 5 c.c. into the skin of the flank. Chloral and bromide, āā gr. iij *quartis horis,* were administered by the mouth. Temperature on admission 97° F. and in the evening 101° F. Feeding by nasal tube inducing spasms he was given Slinger's meat suppositories by the bowel. At 8.50 p.m. he had another spasm, got very blue, and suddenly expired.

At the post-mortem nothing was found but cerebral congestion, which was very marked. The brain was very soft and diffluent to the touch. The right side of the heart was congested, as also the liver. Nothing else abnormal was observed.

(*Read April the* 10*th*, 1908.)

Discussion.

The Chairman (Dr. Edmund Hobhouse) (Brighton) said he had no experience of the disease, but believed it was chiefly associated with the Island of St. Kilda, where it was at one time very prevalent. It was common in tropical climates, and was doubtless infective.

Dr. Edmund Cautley said he had a case of similar kind two or three years ago. The symptoms began on the fifth day of life, and the child died on the ninth. If the disease commenced later there seemed to be a better chance of recovery. In consequence of that, undue importance was attached to treatment. He did not think any of the sera had been of great value in the condition. The injection of a 25 per cent. solution of magnesium sulphate into the subdural space had been said to do good; so, too, the subcutaneous injection of rabbit's brain, though the latter caused local abscesses; still, a Viennese doctor claimed to have cured thirteen cases out of sixteen by that means. Probably a more common cause of the condition than Fuller's earth was charred brown paper, which many people used. That was made out of the worst possible refuse, and was likely to contain the tetanus bacillus. He asked what treatment was used in the cases. Chloral given very freely might produce good results; it should be given by the rectum. For infantile spasms in early life it was even more satisfactory than bromide, but the two drugs could be combined. Recently in a case of sepsis neonatorum, with a temperature of 103°–105° F., chloral was the only drug which seemed to control the spasms effectively.

Dr. G. A. Sutherland said the last case of the kind he had was in a child, aged 1 month, who had convulsions, and had been ill from the second week of life. It was found that the navel had been dressed with Fuller's earth. From the vaginal discharge an organism was cultivated closely resembling the tetanus bacillus. There was opisthotonos and pleurothotonos, and a number of medullary disturbances. The respirations were 120 per minute—the most rapid he remembered in a child. Eventually the child died, and it was found, post-mortem, to be a case of non-development of the cerebrum. The brain was the size of an ordinary potato, with all the convolutions extremely atrophied except at the posterior part of the occipital lobes. The cerebellum was normal. The symptoms during life had been extremely suggestive of tetanus.

A Member said he had a large practice in confinements among the poorer classes, and Fuller's earth was used by probably 95 per cent. of mothers. To attribute the disease to that was, he thought, casting an undeserved onus on the preparation, which was not more harmful than ordinary toilet powder.

Dr. Harry Campbell said it would be interesting to know whether, in Dr. Sutherland's case, the cerebellum was absent, because there was a condition which Sherington had called attention to in which, when the cerebellum was removed, rigidity of muscles was produced. He suggested that possibly Dr. Sutherland's case was an example of this so-called "decerebrate rigidity."

Dr. George Carpenter said that one successful case of treatment by anti-tetanus serum had been reported to the Society by Dr. J. McCaw, of Belfast.* He agreed as to the value of chloral and bromide in ordinary convulsions, but in tetanus neonatorum they seemed to be valueless.

Dr. Porter Parkinson, in reply, said he had no experience of the treatment of the condition by magnesium sulphate or rabbit's brain. He ordered large rectal injections of chloral and bromide, *i. e.* 3 and 5 gr. respectively every four hours for a time, but through a misunderstanding on the part of the house-physician it was only given once. But three days later it was resumed, and pushed, but he did not see any marked benefit from it. The spasms were fairly continuous, and were not increased by feeding or examination of the child; there was very little exacerbation. Dr. Carpenter's cases seemed to be much like his own. He knew nothing of treatment by serum, but he looked up the matter in Holt's book, and he found that nearly all the cases which recovered did so when they were taking chloral and bromide. Dr. Sutherland's likeness of the size of the brain to a potato did not give much idea, as those vegetables varied so much in size.

76. CASE ILLUSTRATING EUSTACE SMITH'S BRUIT.

By LEONARD GUTHRIE, M.D.

The patient is a healthy-looking boy, aged 7 years. Since contracting measles last November he has suffered from debility, spasmodic cough, and wasting. He has had frequent attacks of bronchitis. There is no family history of tuberculosis.

When first seen January the 30th, 1908, the superficial veins in front of the chest and shoulders and the temporal veins were prominent and evidently engorged, especially on the right side.

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* 'Reports,' vol. i, p. 102.

On auscultation a musical undulating venous murmur could be heard chiefly in the second right intercostal space. The bruit occupied the whole of the cardiac cycle.

The heart-sounds are normal, and the heart not enlarged. Percussion note was diminished in resonance over the upper part of the sternum and in the upper two intercostal spaces to right and left of the sternum.

The venous bruit was greatly intensified on retracting the head.

Since admission the physical signs have become much less pronounced. The veins on the front of the chest are less noticeable than they were. The venous bruit is fainter and divided into systolic and diastolic until the head is retracted; it then resumes its undulating musical character, is much louder and continuous. After retracting the head for a few moments the superficial veins again stand out prominently.

Eustace Smith, who first described this form of venous bruit, attributed it to enlargement of the mediastinal glands and of the gland at the bifurcation of the trachea in particular. He believed that retraction of the head in such cases tilted the trachea forwards with the enlarged glands, which pressed upon the innominate or superior vena cava, and thus gave rise to the characteristic venous hum.

The diagnostic value of the sign has been questioned, but in cases like the present in which venous obstruction is obvious there can be little doubt as to the presence of mediastinal growth or enlarged glands. A similar bruit, however, is common enough in anæmia, and so taken alone is no proof of the existence of mediastinal growths or enlarged glands.

A point of interest which, as in the present case, is often observed, is that the physical signs are very variable and may subside altogether. Probably in such cases mediastinal glands become temporarily engorged and enlarged, and so give rise to the condition described.

The glands most frequently involved by tubercle are, as Dr. F. E. Barton has pointed out, seen in the neighbourhood of the right bronchus at the bifurcation of the trachea and at the root of the right lung. In such cases piping and almost tracheal breath-sounds may be heard over the right bronchus back and front. A spasmodic cough much resembling whooping-cough is often a marked feature.

In many cases tuberculous mediastinal glands shrink and become calcified and do no harm. But they may ulcerate into a bronchus or into a large vessel causing fatal hæmorrhage, or they may give rise to mediastinal abscesses or be the origin of a general spread of miliary tuberculosis.

Most cases of tuberculous meningitis are associated with caseating mediastinal or bronchial glands.

(*Exhibited April the* 10*th*, 1908.)

77. A CASE WITH EUSTACE SMITH'S BRUIT.

By F. LANGMEAD, M.D.

The patient is a girl, aged 5½ years, who has never been quite well since she had whooping-cough in April, 1907. Since then she has been the subject of frequent coughs and colds. The cough is neither laryngeal nor paroxysmal. She is rather pale, but does not look particularly ill. On examination of the chest there is some impairment of note in the inter-vertebral region of the right side. There is nothing more definite to indicate pulmonary disease. To the right of the manubrium sterni a definite to-and-fro rushing murmur is heard if the head is held retracted, but is lost when the neck is flexed. The bruit is such as was described by Dr. Eustace Smith as associated with enlarged bronchial glands, and is probably venous in origin.

There is no family history of tubercle, and the patient is the only child. (*Exhibited April the 10th*, 1908.)

Discussion.

The Chairman (Dr. Edmund Hobhouse) (Brighton) said he thought there was still some dulness over the manubrium sterni in Dr. Guthrie's case. From the history he had little doubt it was a case of enlarged glands.

Dr. Edmund Cautley said he thought Dr. Guthrie took up the right position as to the value of the murmur, namely, that it was of very little importance apart from other physical signs, notably dulness and dilated veins. He would go even further and maintain it was of no diagnostic value. It was important to decide authoritatively, for children were condemned to a life of invalidism because they had the so-called "Eustace Smith" murmur, and were therefore considered to have tuberculous mediastinal glands. He had seen many children with that murmur who had not developed other signs of tuberculous glands. Some of them he had had under observation many years. Two he could recall now, and he still occasionally saw them. In one the murmur was more marked when the child became anæmic, but neither of them had developed anything to suggest tuberculosis.

Dr. Porter Parkinson said he was in absolute accord with Dr. Cautley; he had kept his eye upon several cases in which that murmur was heard, and he did not know that anything serious had developed in any of them. He did not think the murmur, without any other obvious evidence, was of any importance. He would be glad if the Society, some time, would discuss what the signs of enlarged glands in the chest were. In many cases enlarged mediastinal glands were present without any signs pointing to their existence.

Dr. F. W. Higgs asked what type of cough there was. He thought the most important sign of enlargement of bronchial glands was a spasmodic cough, very much like that of whooping-cough. He had diagnosed the condition several times, and the only case which he had traced to the post-mortem room was correctly diagnosed; and there was no Eustace Smith murmur in that case. He asked whether Dr. Guthrie had had a skiagram taken of the thorax. He, Dr. Higgs, thought he found some impairment of resonance not only over the sternum but on the left side of it. Also, did Dr. Guthrie think it was usual in tuberculosis of bronchial glands to find venous obstruction? He had never seen it in the cases he had met. Possibly in the present case the intra-thoracic glands were affected with new growth and not tuberculosis.

Dr. George Carpenter said he thought the enlarged veins in the neck were of diagnostic importance. But the Eustace Smith bruit might occur with or without organic changes. He had also met with the bruit in connection with four different types of cases: (1) in pericarditis with mediastinitis; (2) with sarcoma of the mediastinum; (3) with tuberculous growth in the mediastinum; (4) with abscess of the thymus. With regard to the case of tuberculous growth in the mediastinum, that subsequently was associated with œdema of the left hand and arm, and, post-mortem, it was found to be due to blocking of the corresponding innominate vein. He did not attach any special diagnostic importance to Eustace Smith's murmur. He had not had sufficient experience to speak dogmatically as to the value of X-ray examinations in such cases, but Variot, of Paris, claimed very useful information from this practice.

Dr. Leonard Guthrie, in reply, said members seemed agreed as to the diagnostic value of the murmur. He did not think it was of any value unless there were other physical signs to back it up. He, Dr. Guthrie, had almost come to believe he could diagnose the presence of enlarged glands by other physical signs, chiefly the diminution of resonance, which was usually to the right of the sternum, in the second and third spaces, and secondly by the character of the breath-sounds. One had to recognise that there was a physiological harshness over the interspaces on the right side. But one found more than that: the breath-sounds were almost tracheal in character, and were heard behind as well as in front. He agreed that the spasmodic cough was a very prominent symptom in such cases, and it was frequently regarded as whooping-cough. Yet there was rarely any vomiting and the whoop itself was a very half-hearted affair compared with the genuine article. He would have an X-ray examination of the chest made, but he was not confident that it would discover anything which could not be ascertained without. He remembered Dr. Eustace Smith explaining the formation of the bruit in the way he, Dr. Guthrie, had described, but he said it was not produced in cases of enlargement of the thymus, because the thymus was in front of the vein, whereas the gland was behind it, and when the head was thrown back the trachea and its enlarged glands became tilted against the vein.

78. A CASE OF AORTIC REGURGITATION IN A BOY AGED FOUR YEARS.

By J. PORTER PARKINSON, M.D.

Leslie B— was brought to the Hospital for "fainting fits." He had a healthy family and personal history, and was said to have had no pains, or other evidence of rheumatism, or other acute specific sore throats.

On admission he was well nourished, but somewhat anæmic. The cardiac impulse was in the fifth space just outside the nipple line. At the base of the heart was a well-marked diastolic murmur, best heard on the left side and conducted down the sternum. The pulse was 94, regular, and collapsing in character. Capillary pulsation was well-marked. The other organs were normal.

Unfortunately, to-day, perhaps owing to the excitement of this meeting the murmur has disappeared, but the character of the pulse is evident.

The case is shown to elicit the views of the members of this Society as to the ætiology of the murmur, and as to whether they consider it likely to be of congenital origin. (*Exhibited April the* 10*th*, 1908.)

Discussion.

The Chairman (Dr. Edmund Hobhouse) (Brighton) said he listened to the child, but, in common with others, he had failed to hear the murmur which Dr. Parkinson said existed. He did not know what the causation could be in a child of that age. Of course members had seen cases in which a murmur appeared and disappeared.

A Member said he listened to the boy's heart just before entering the room, and heard the murmur distinctly.

Dr. Edmund Cautley said he had had an adult with an aortic diastolic murmur from rheumatic endocarditis in hospital. The murmur disappeared and remained absent for a week or ten days. He formed the impression that the case was cured, and showed it as such, but the murmur returned during an attack of mumps while the patient was still under treatment.

79. A CASE OF ENCEPHALITIS; BROMIDE RASH FOLLOWING A DOSE OF TWO AND A HALF GRAINS OF BROMIDE OF POTASSIUM.

By GEORGE CARPENTER, M.D.

Florence C—, aged 20 months, was admitted into the Queen's Hospital for Children on February the 10th, 1908. Four weeks previous she was taken ill with measles and had not been well since. "Months before" she had whooping-cough and "the cough seemed to have returned while she had measles." About a week after the measles she had bronchitis. Dr. E. Henderson sent her to the hospital with the account that she had "frequent cerebral cry with squinting, but there was no retraction of the head or vomiting." She had screamed since February the 3rd; a squint was first noticed on that date, and her mother thinks she had a fit at that time; she also awoke from sleep screaming. The parents had four children living and two deceased; one of the latter died, aged 5 years, of tumour of the brain, the other, aged 18 months, of scarlet fever and convulsions "like this child."

When admitted into hospital there was no evident retraction of the head, and the neck muscles were not stiff and the abdomen was not obviously retracted. There was no rigidity of the legs, the knee-jerks were not obtained and no Babinski either. She cried when touched, and was very feverish and ground her teeth at times. She had definite double optic neuritis of meningeal type. There was "some squint," but the notes do not state what muscles were affected. Temperature 98° F., pulse 118 to the minute, respirations 24. On February the 14th the temperature was still subnormal. There was a little pus in the urine, and the weight was 1 stone 4¼ lbs. On the 16th bromide of potassium mixture ʒj thrice daily (5 gr. ad. ʒij) was ordered; after a dose of 2½ grains of the drug a typical bromide eruption appeared. Some lesions disappeared, but some grew steadily and became umbilicated and others became fleshy.

The dose was repeated thrice daily for two days and then stopped, since when she had an occasional dose of 5 gr. of bromide of potassium and 2 gr. of chloral hydrate.

On the 22nd the weight had fallen to 1 st. 1¾ lbs. She was given an injection of tuberculin 0·002 mgrm. The temperature was still subnormal, and it was subsequently unaffected by the injection.

On the 25th she had a discharge of pus from the right ear; the ear was syringed, but with no relief of symptoms.

A note on the 27th states that there was still blurring of the discs.

March the 4th.—The same constant screaming, like a meningitic cry. The temperature continues subnormal.

March the 5th.—Calmette applied to the left eye; no reaction. Pulse 96–120 per minute, respirations 24.

March the 9th.—The right optic disc was a trifle pale, and one of the veins was just a trifle heightened in colour as it crossed the disc edge; the blurring had gone. The left disc also was a trifle pale but otherwise unaffected. On this date there was a bromide eruption, chiefly on the legs.

March the 20th.—Bromide eruption gradually subsiding. Sits up naturally now, but is very irritable and cries incessantly at times for no apparent reason. Temperature still subnormal.

March the 22nd.—Lumbar puncture performed; the fluid spurted out, being under considerable pressure. There were no cells in it. The microscopical examination was abortive owing to contamination. It was not sterile. She was considerably quieter after the puncture.

March the 28th.—Lumbar puncture again performed, and the fluid was centrifuged and examined immediately afterwards. Neither cells nor organisms seen. There was not so much pressure on this occasion. She is much quieter and doesn't cry so much.

April the 2nd.—Bromide rash has disappeared, pigmentation alone being left. She cries without apparent reason, and the cry appears to be lacking in intelligence. She stops when fed.

April the 5th.—The child seems to be quite happy and is much quieter, but she does not appear to be intellectually fit. She has had bromide occasionally, but has experienced no return of the rash. The knee-jerks still could not be obtained. Has increased in weight of late 1¼ lbs. since March the 20th.

Remarks.—The child appears to have had an attack of encephalitis following measles, the brunt of which has fallen upon the psychical areas of the brain. What the future may have in store for this child is not clear, but the possibility of her moral and intellectual nature being adversely affected by the illness is one that cannot be ignored in view of her present unsatisfactory mental condition. It is not unlikely that she will pass into a state of moral and intellectual imbecility, but to what extent the future alone can tell.

(*Exhibited April the 10th,* 1908.)

Discussion.

The Chairman (Dr. Edmund Hobhouse) (Brighton) thought the discs were pale. The appearance of the bromide rash after only 2½ grains was very interesting, though infants were known to be very susceptible to the drug. Lumbar puncture was of interest in such cases. In many cases in which lumbar puncture was performed meningitis was suspected and the symptoms disappeared; in one case in which there was marked retraction of the head the symptoms disappeared after it. It came on after pneumonia, and there were pneumococci in the fluid. Lumbar puncture he thought might be made use of in doubtful cases with advantage.

Dr. George Pernet said a bromide rash might appear after a very small dose of the drug, though 2½ grains seemed a small quantity to bring out such a rash as had been described, but it was not impossible. Perhaps the child had been having bromide outside the hospital. Sometimes he had diagnosed bromide rash in the face of stout denial that any had been taken. In one case the general opinion favoured syphilitic eruption in a baby, but he maintained that it was bromide eruption. Eventually, after a lot of trouble, the mother admitted she was an epileptic, had been taking bromide of potassium, and had been suckling the child. Many hospital patients tried a number of things before going to the hospital. Children were often suckled for long periods. In a case (not bromide) he had hoped to show that afternoon the mother was still suckling her baby of eighteen months.

Dr. George Carpenter, in reply, said the case was not suckled in the hospital. Bromide was given for the first time six days after its admission. He had met with as typical a case as that narrated by Dr. Pernet. A baby in arms had on its face a most unmistakable bromide eruption, and he had tried to find the cause for it, but the mother would not admit that she was taking any drugs, and she stoutly asserted that her baby had not been given any; nevertheless the baby had had bromide at some time or other, of that he was certain.

Postscript, August the 13*th*, 1908.

In July last she was re-admitted into hospital to have lumbar puncture performed; the fluid was sterile. To-day, by request, her mother brought her for examination. She stated that she was quite as bright and intelligent as other children, but said that she occasionally had had screaming fits and bit her playmates. She talks and walks well. Her mother asserts that she is more observant than other children of her own age—*i.e.* 2 years—and that she knows the different animals. She asks for all she wants and she sleeps well, but her appetite is poor. I examined her fundi oculorum and found the optic discs perfectly normal; in colour they were a shade or two lighter than the red reflex. There were no physical signs.

80. INFECTIVE ENDOCARDITIS OF PULMONARY VALVE COMMUNICATING WITH THE AORTA.

By EDMUND HOBHOUSE, M.D.

Heart from a child, aged 7 years, with infective endocarditis of the pulmonary valve, probably supervening on congenital disease. This has ulcerated through and communicates with the aorta. There was a very loud, harsh, and almost continuous bruit during life.

(*Exhibited April the* 10*th*, 1908.)

81. STOMACH FROM A CASE OF TETANUS.

By EDMUND HOBHOUSE, M.D.

Stomach of a child, aged 3½ years, who died of tetany. The stomach is very small, and there was no dilatation of the colon. (*Exhibited April the* 10*th*, 1908.)

82. BILATERAL RENAL APOPLEXY.

By EDMUND CAUTLEY, M.D.

A male infant, aged 3 months, was admitted into the Belgrave Hospital for Children on March the 26th, 1908, for convulsions, from which he died two days later.

For two months he had attended the Out-Patient Department. When first seen he was wasting and had pemphigus. His weight was 5 lbs. 14 oz. In addition he presented an unusual deformity of the right hand, the second and third fingers being flexed at the proximal inter-phalangeal joints by thickened bands of fascia, analogous to what is seen in Dupuytren's contracture.

Up to March the 16th he steadily improved and increased in weight to 8 lbs. 7 oz. On the 26th at 2 a.m. he developed convulsive movements of the limbs, refused food, and became drowsy. After this he never properly became conscious. On admission he was collapsed, with cold extremities and sunken fontanelle. Twitching movements and a few mild general convulsions continued until death, fifty-three hours after the onset. The spasms chiefly affected the lips and face, and, to a less extent, the hands and arms. Nystagmus was sometimes present.

The temperature was subnormal on admission, rose to 102° F. at night, steadily fell to below 98° F. next evening, and reached 100·4° F. before death.

The pulse-rate and breathing were very rapid. There was no vomiting but some difficulty in swallowing, and the stools contained curds.

The child was the third in the family, born at full time, normal labour, and breast fed. The mother had had no miscarriages. Her first child was stillborn.

Post-mortem.—Nothing abnormal found anywhere except in the kidneys, each of which showed renal apoplexy to the extent of one half to two thirds of their substance.

The thymus was unduly large.

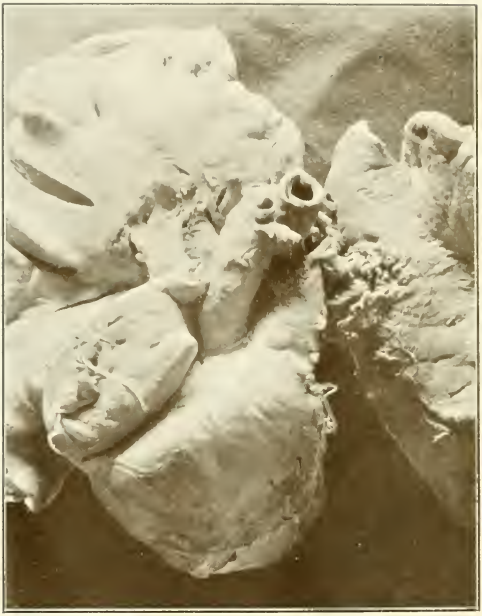
(*Exhibited April the 10th*, 1908.)

Discussion.

Dr. George Carpenter asked whether Dr. Cautley suspected syphilis in the case. A similar condition of kidney had been reported by Fruhinsholz in a syphilitic infant. There was a possibility that syphilis was present in the case, especially as there was pemphigus. Perhaps Dr. Cautley would have the spirochæte examined for and settle that point. If it could not be found, syphilis was put out of court, and there must be some other cause—bacterial emboli, for example, as Czerny and Moser have demonstrated. It might be explained on the assumption that the eruption was the bullous variety of impetigo contagiosa. Thrombosis of the renal veins does occasionally complicate the nephritis associated with gastro-intestinal catarrh in infants.

Dr. George Pernet asked whether the bullæ were on the palms and soles or scattered about the body. Also what form

PLATE VIII.



Heart and lungs, natural size, removed from a Mongol aged 5 months. Note the aorta passing over right bronchus. To the left is the superior vena cava, behind this the pulmonary vein, and behind that the right branch of the pulmonary artery. On the right side is the pulmonary artery with the ductus arteriosus passing over the left bronchus. To the right of this in the foreground is a cut pulmonary vein, behind that the left bronchus, and above the bronchus is the left branch of the pulmonary artery. Note the windpipe cut across, behind this the œsophagus with a stick in it. The aorta is seen with the vessels for the upper extremities arising from it. (Photograph by the author. Natural size.)

Dr. George Carpenter's Case of Congenital Malformation of the Heart.

Adlard & Son. Impr.

of pemphigus it was? Was it due, perhaps, to general infection? Of course it might have been a pemphigus neonatorum and not of a syphilitic nature at all.

Dr. Edmund Cautley, in reply, said he did not suspect syphilis, and there was no reliable evidence in favour thereof. The child was the third in the family, and was born at full term. The first child was still-born, but there had been no miscarriages. Although the child weighed under 6 lbs. at a month old, it got on remarkably well. He did not know what the rash was like, and he had only the out-patient note to refer to. From that he concluded it must have been mild pemphigus, and it occurred two months before the final illness. The child was under observation in the hospital two days, and nothing wrong was noticed in the urine. The ward sister was an excellent observer, and if there had been scanty micturition or staining of the napkin it would have been reported.

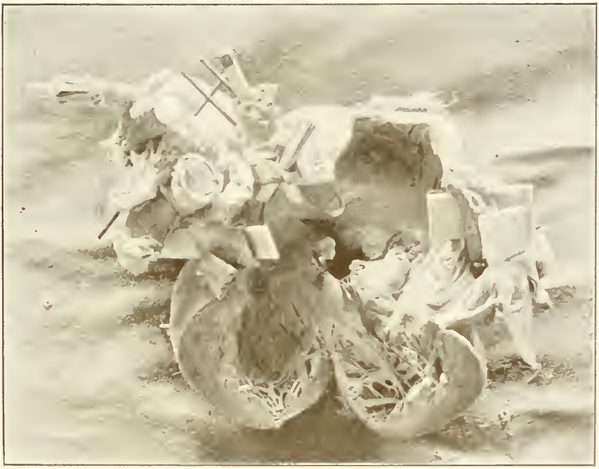
83. CONGENITAL MALFORMATION OF THE HEART; HEART OF THREE CHAMBERS (ONE AURICLE AND TWO VENTRICLES), SEPARATED BY A LARGE BICUSPID VALVE; AORTA PASSING OVER RIGHT BRONCHUS, PATENT DUCTUS ARTERIOSUS OVER LEFT.

By GEORGE CARPENTER, M.D.

The specimen was removed from a Mongol, aged 5 months, which was shown at the meeting on March the 20th, No. 63, p. 278.

The arch of the aorta is seen passing over the right bronchus (*vide* Pl. VIII), and a small patent ductus arteriosus passes to the left of the trachea and the œsophagus—which is marked by a piece of wood in the tube—and joins the aorta on the left. The aorta and the pulmonary artery possess the normal number of valves and are of natural proportions. On the right side will be seen (left of picture) the superior vena cava entering the right auricle, and behind it is a large pulmonary vein, and behind and above that the right division of the pulmonary artery. On the left side (right of picture) is first a pulmonary vein cut across displaying behind it the left bronchus below, and just above the bronchus the left branch of the pulmonary artery. The aorta is seen to be giving off four separate trunks in the position of the brachio-cephalic vessels, the fourth being in close proximity to the ductus arteriosus. In Pl. IX the heart has been separated from the lungs. A cut has been made vertically through the auricle and ventricle laterally, and the organ has been opened out and pinned in position. This section displays above, an auricle of only one chamber without a trace of septum. The right side is large and fleshy (*vide* Pl. IX also) with prominent columnæ carneæ, the left, which is not specially noticeable in the picture (Pl. IX), is small and membranous. At the top left-hand corner is the superior vena cava—a hair-pin has been passed through that into the auricle (*vide* Pl. IX also). In front of this are two veins (right pulmonary) with pieces of wood passing through them into the auricle. Below this to the left is a piece of a large vein (left pulmonary) passing into the auricle, which here is membranous. It also has a piece of wood in its channel. On the right of the auricle is the opening of a piece of the inferior vena cava passing into the fleshy auricle with a stick of wood in its channel. In front of that is a piece of wood which marks the track of one of the left pulmonary veins passing through the membranous part of the auricle. The top of the inter-ventricular septum is well seen, and above it is a bicuspid valve of considerable size separating the auricle from the ventricles and possessing tendinous attachments to the papillary muscles of both ventricles. On the right side there are some filamentous strands partially attaching the cusp to the ventricular septum, but on the left these are absent. The front valve has been injured in preparation, and is seen torn across in the far left-hand corner, but during life the parts were united. The valve is of a dull leaden colour—of morbus cæruleus hue even after soaking for weeks in

PLATE IX.



Heart of a Mongol aged 5 months. The left side of the heart has been opened by a vertical slit and displays the following features: One auricle: fused mitral and tricuspid valves making one large bicuspid valve; the top of the ventricular septum. For other features see text. (Photograph by the author. Natural size.)

Dr. George Carpenter's Case of Congenital Malformation of the Heart.

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formalin. There is a distinct interval between the under surface of the valve and the top of the ventricular septum, more obvious on the left side than on the right because the belly of the right cusp (posterior) hides it, so that during life there must have been a communication between the two ventricles. On the left of the cardiac chambers will be noticed the following structures. On the extreme left is the descending aorta with a piece of wood in it. In front of that is the œsophagus with a round-headed pin within it. Next are the right and left bronchi with a piece of wire passed through them, and next the branches of the pulmonary artery filled with blood-clot. To the left of the aorta, seen as a rounded cord, is the ductus arteriosus, passing from the former to the pulmonary artery; its junction is obscured by the left bronchus. All these structures have been left *in situ*, but they are only called attention to merely as a matter of passing interest. The important features of this figure are those which have been previously described.

(*Exhibited April the* 10*th*, 1908.)

Discussion.

The Chairman (**Dr. Edmund Hobhouse**) (Brighton) asked whether there was any cyanosis. It showed very well one of the protean aspects of mal-development of the heart. As Dr. Carpenter had remarked, most prudent men did not care to diagnose such conditions.

Dr. George Carpenter, in reply, said there was some cyanosis when she was admitted into hospital, but that disappeared while there, although occasionally she had attacks of it. But although cyanosis was by no means a feature of the case there was a free mixture of venous and arterial blood.

84. TRAUMATIC SEPARATION OF THE LOWER EPIPHYSIS OF THE FEMUR.

By JOHN POLAND.

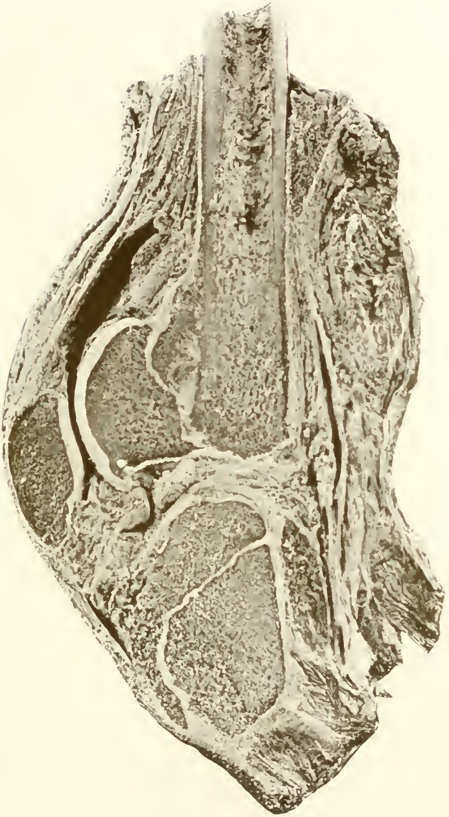
This specimen (Pl. X) was from a well-grown and good-conditioned boy, aged 14 years.

The accident was a violent blow on the front of the left knee from the handle of a grocer's cart, which knocked him down. In falling his leg was much twisted.

The patient was able to get up and was treated by a doctor with a side-splint. Although a bruise was noticed on the outer side of the knee nothing serious in the way of displacement was made out, no crepitus, etc., but on the third day great tenderness became very noticeable all round the knee-joint with effusion into the articulation, yet without any rise in general temperature. These signs subsided gradually until he was admitted into the hospital on the twenty-sixth day, with great thickening and swelling about the joint and lower end of the femur. The joint was then held somewhat flexed, and passive flexion could only be effected through 48°. On the outer side a bony projection could be detected. The patella was freely movable and the knee-joint could be completely extended.

The skiagram (Pl. XI), which I also show, revealed the fact that the lower epiphysis of the femur was completely displaced *forwards* with the patella. The somewhat *outward* displacement of the diaphysis had evidently saved its backward displacement from doing any injury to the popliteal vessels, which is so commonly the result of this displacement in these separations. This decided that an operation for detaching the epiphysis from its connections with the front of the femur and replacing it in position was imperative. However, after the first incision the patient collapsed from the anæsthetic and succumbed before anything further could be done. The specimen

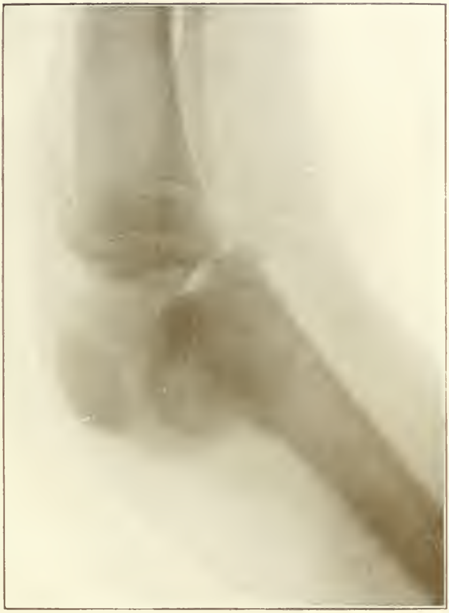
PLATE X.



Mr. John Poland's Case of Traumatic Separation of the Lower Epiphysis of the Femur with Displacement Forwards.

Adlard & Son, Impr.

PLATE XI.



Skiagram of Mr. John Poland's Case of Traumatic Separation of the Lower Epiphysis of the Femur with Displacement Forwards.

Adlard & Son. Impr.

has, therefore, an unusual interest to me inasmuch as the case is the first and only one I have lost under an anæsthetic during the whole of my surgical work.

The vertical section of the specimen displays the appearance of this epiphysial separation after the lapse of only thirty-eight days; it is therefore unique as far as museum specimens are concerned.

The deposit of soft callus beneath the separated periosteum in front, that is, between it and the diaphysis, is especially to be noted, but the other appearances connected with the backward and outward displacement of the diaphysis, and escape from injury of the popliteal vessels, are equally well displayed as in other cases previously recorded. (*Exhibited April the* 10*th*, 1908.)

85. THE "EMPTY BRONCHUS" TREATMENT BY POSTURE IN THE BRONCHIECTASIS OF CHILDREN.

By WILLIAM EWART, M.D.

I do not propose on this occasion to go over the entire ground which was recently covered by my paper on "The Principles of Treatment of Bronchiectasis," read before the East Sussex Medico-Chirurgical Society at Hastings, but rather to confine myself to the special points in the mechanical part of the treatment in children. This intractable disease, if it is to be cured at all, is more likely to be cured in childhood than at any other age. And the responsibility is great of not missing that better chance by delay.

A sad instance in point is the lamented death a few days ago of the little patient, W. C—, aged five years, who had suffered from the affection since an attack of pneumonia nearly three years ago. Her systematic treatment in July, 1907, on the inclined bed, and by the prone posture on the all-fours exerciser, was of very short duration owing to the temporary closure of the ward. When at last her mother brought her again a month ago, she had developed lardaceous disease and albuminuria. Although after a few days of postural treatment the expectoration was greatly reduced, so that even partial inversion did not lead to much being coughed up, the fœtor showed no improvement ; and the probability that ulcerative and necrotic changes had occurred in the dilatations was borne out by the patient's rapid failure and final exhaustion ; but an autopsy could not, unfortunately, be obtained. I believe that had she been under continuous treatment during the last twelve months she would have been, not only alive to-day, but probably vastly improved under the treatment which I have to describe to you as the "empty bronchus method." For there is reason to believe that a well-drained bronchus is also one that practically ceases to secrete.

The principles of treatment.—This brings me to say a word of introduction on the general question. There are two fundamental principles which must be carried out if any line of treatment which may be adopted is to be successful. One of them is to *empty the bronchus, and to keep it empty.* The other principle is to *fill the lung with air, and to keep it actively expanding.* These two objects were conveyed in the title of the paper published in the 'Lancet' for July the 13th, 1901, which tells of the difference between the practice of this century and that of previous ones—"The Treatment of Bronchiectasis by Posture and by Respiratory Exercises."

The various modes of treatment.—On this as a basis various therapeutic measures may be founded with good hope of success. It is not my object to deal with them to-day, but it is right to enumerate them. (1) The latest, which is advocated on rational lines, and practised with success in phthisis as well as in bronchiectasis by Dr. Colin Campbell, of Southport, is the direct medicinal treatment of the bronchus by *intra-tracheal injections.* He finds that it is capable of application even in young children. (2) Of great importance is also Arnold Chaplin's well-known method of *creasote inhalation.* It is also applicable in young children, and it is recognised to be the most effectual means of overcoming by medicinal agencies the fœtor of the expectoration and of the breath. (3) In the third place, we have a variety of internal remedies which I need not enumerate; and, lastly, (4) a large assortment of inhalations made available by methods to which I may presently refer.

The mechanical methods which I have to urge as essential adjuncts to these various plans combine two objects, *the bronchial and the pulmonary*, which are distinct, but possess one great feature in common—they are both intended for *continuous application*, in the strict sense.

THE INTERMITTENT POSTURAL TREATMENT.

The bronchial treatment is mainly postural. It is strange that the obviousness of this indication should have so long escaped notice. My own earliest employment of the *ultra-prone position or inversion* dates back many years in connection with the emptying of basic cavities in phthisis. It was not a novel device, at any rate in this country, and had been advocated and practised before; and it is the type of what I shall term the *intermittent postural method*, which should rightly be connected with the name of H. Quincke,* of Kiel. In 1898 he published his short but weighty "Contribution to the Treatment of Bronchitis," in which he suggested the inclined prone position for adoption for comparatively short periods once or twice daily, for the sake of bronchial drainage in cases where this was wanted. In October, 1900, Jacobson† published a further account of the method, with cases in which the inclined posture had been used for periods of one hour once or twice in the course of twenty-four hours with favourable results. These papers were not known to me until I consulted the literature in connection with the publication of my own paper mentioned above. In the latter I advocated the *continuous postural method* by keeping the patient in bed, and by raising the foot of the bed considerably so as to keep up a strong downward slant from the bronchi to the larynx, whatever the positions might be into which the patient might turn during sleep.

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* 'Berlin Klin. Woch.,' 1898, p. 525.

† *Ibid.*, 1900, p. 904.

The intermittent "ultra-prone" posture or "inversion" is the most rapid and effectual for the evacuation of accumulations in the cavities of phthisis or of bronchiectasis. The patient leans over the side of the bed or of a table, placing the hands on the ground, so that the long axis of the thorax approaches to the vertical. A few coughs will rapidly clear away the greater part of the collection. The method is fortunately available in children in a degree not to be attained in the adult, for even up to adolescence they are not alarmed at the thought of "standing on their head." Infants will bear to be held vertically head downwards, and in small children something approaching to actual inversion may be safely resorted to. These extreme positions, in which patients are encouraged to cough and to expectorate, are exceedingly useful for dislodging remote accumulations, and it may be possible to improve the opportunity by the judicious application of bi-manual pressure at the base of the thorax judiciously performed and limited to the period of expiration. As a fact, *absolute* inversion is not necessary. The child may be either held in the ultra-prone position by the nurse, or laid across a low chair or a stool, with the hands on the floor, in imitation of the procedure in the adult.

The intermittent prone postural method of Quincke is likely to come into extensive use in various affections, and I have devised with that view, and had constructed, a set of appliances or machines enabling me to apply the prone method with graduated inclinations and lateral positions, according to the requirements of the case and of the affection under treatment.

THE CONTINUOUS POSTURAL TREATMENT.

The continuous treatment has three practical instalments: *The nocturnal postural treatment; the diurnal postural treatment;* and the *respiratory treatment by means of the elastic belt and of the "exerciser."*

(1) *The costo-abdominal elastic belt* should be worn during the night as well as the day, for its virtue is active for good during each breath that is drawn. If this were neglected, the loss of advantage to a child during the ten hours' night sleep might be easily reckoned up in figures to an important total. The belt itself, made of waterproof material, easily washed and dried, has its buckles in front, and at one or both sides an insertion of a lacing of indiarubber cord or tubing. It is mainly *abdominal*, extending some way below the umbilicus, and upwards not quite as far as the costal arch. But I have found it useful to attach to it in the back a *lower thoracic elastic band*, one or two inches wide, also buckling in front, which, being quite separate except over the spine, where it is continuous with the belt, can be carried obliquely over the costal arch, and its buckle fastened to the nightgown by a safety-pin at any level desired. Its pressure can thus be regulated nicely so as to be just sufficient to stimulate the costo-abdominal breathing. I have explained elsewhere the probable mechanism of the advantage accruing to the upper costal breathing from wearing the abdominal belt. The recurring stimulation of the diaphragm with each expiration by the artificial elastic recoil of the abdominal contents calls reflexly into action some of the apparatus for extraordinary respiration, and in particular the upper costal muscles, whilst it directly amplifies in a marked degree the inspiratory excursions of the diaphragm. It goes without saying that judgment is requisite in dealing with so active a therapeutic agent.

(2) *The nocturnal treatment.*—This should be made

as effectual as possible. Everything depends upon the declivity of the inclined plane being effective, not, as might happen, illusory. In the first place, as the distance from the diaphragm to the larynx is so short in a little child, *the angle* of the inclined plane must be relatively greater. In the "bronchiectasis cot," which I have to describe, the foot end of the "fracture-bottom" must therefore be secured to its graduated support at a higher level.

The "bronchiectasis cot" is easily prepared in the case of the ordinary bed-shaped cot with high end rails as well as side rails. A fracture-bottom is placed under the mattress, and its foot end must be raised to the desired height by means of the *graduated support*, the other end extending as far as the head rail.

The graduated support is simply a firm board of sufficient width, from which project two thick movable wooden pins to bear the weight of the foot end of the fracture-bottom. The board is perforated with two parallel vertical rows of holes to receive the pins. A strap passed through two of the holes not in use and round the upright rails will ensure its stability. This, or some similar contrivance, will be found convenient for hospitals, as it occupies little space for storage and offers the choice of several positions to suit various cases. For private patients it is easy to obtain the same result by slinging the end of the fracture-bottom on a rope fastened to the foot posts, and passing underneath through eyes or holes to prevent any slipping.

The mattress having thus been duly slanted, it is necessary, in the second place, that the slant of the thorax itself be made a genuine one by posture. It is clear that, let the degree of inclination be what it may, if the child were to lie transversely to the cot the long axis of the chest would be horizontal, in spite of the inclination; and as this almost invariably happens it will be often found necessary to let the child lie between two sandbags, which will also afford assistance against the tendency to slide down the slant "into a heap." In the groove thus formed the child can lie in any decubitus; the more this is varied so much the better.

Another practical point is *the bolster*. The bolster must be *small and firm*, for the support of *the head only*; and to this effect it should be secured to the head rails so as not to be displaced. The ordinary square pillow and squash bolster are quite unsuitable, as they extend under the shoulders and thus modify the inclination in the direction of the horizontal. With these practical precautions the nocturnal instalment of treatment becomes one of the most important.

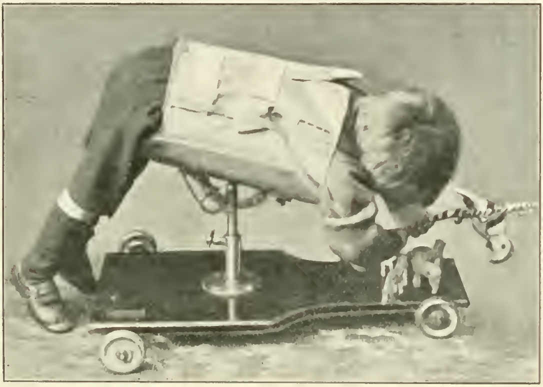

FIG. 58.—The all-fours exerciser in use.

(3) *The diurnal treatment* is, as previously stated, the agent *par excellence* of pulmonary regeneration, whilst it also affords the most effectual means, next to inversion, for drainage. The prone posture which is to be kept up is anatomically the most direct for that purpose (the greater bulk of the lung being posterior), and brings to bear the respiratory activities of exercise for emptying

the sacculations by lateral compression. The all-fours exerciser, upon which the prone treatment is conducted, is a small edition of one of the arrangements which are provided for in my "convalescent machine" for the adult. The supporting body trough, upon which the child is secured by a broad belt, is provided with three sets of movements regulating its height and its inclinations from end-to-end and from side-to-side. The lateral inclination is usually not called into play here. It is specially used for the lateralisation of the chest in empyema, pleurisy, and other conditions requiring special unilateral measures of rest or of re-expansion.

The end-to-end declivity required is that which will bring the shoulders appreciably lower than the hips, and at that angle the child is made to recline in the prone position.

Prone exercising.—The height of the body trough is to be so adjusted as to allow free use of the four limbs—the feet for propelling the machine (the front wheels of which might be made also workable by the hands), the arms and hands for any appropriate work or amusement. In this way, quite independently of any of the dumb-bell and *respiratory exercises* for practice at stated intervals, a good deal of activity is thrown upon the limbs and the neck, and this brings fully into requisition the posterior and basic pulmonary districts. A table can be improvised in front of convenient height for drawing or writing, and to hold the book for reading, or cushions to rest the head. *Prone exercising* need not, therefore, imply idleness or complete interruption of the lessons.

"*The all-fours exerciser*" for children,* and the "*convalescent machine*" for adults,† illustrations of which are included in my paper "On Convalescence and its Stages," ‡ are purposely adapted for most varied medical and surgical affections. They are therefore specially suitable for hospitals and institutions, where they can be in constant use, thanks to their adjustability. But for a makeshift, and as only one position is required, any individual case of bronchiectasis in a child is easily extemporised by padding a high stool, cutting down its legs to the extent desired, and finally mounting it upon wheels. Or we may adapt for our purpose, with slight modifications, Dr. Tucker Wise's "inclined plane for phthisis," a description of which was published in the 'Lancet,' vol. i, p. 1546, 1908.

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* Constructed for me by Mr. G. P. Turner, of 16, Eccleston Street, S.W.

† Constructed for me by Mr. John Ward, of 246, Tottenham Court Road, W.

‡ 'Medical Press and Circular,' February the 19th, 1908.

The rationale of the "empty bronchus method" has probably been made clear by the foregoing practical description. It aims at stopping the bronchial irritation and hyper-secretion by bronchial drainage and by pulmonary ventilation, and at reclaiming for active pulmonary expansion the thoracic space usurped by the dilatations and by the fibrous tissue. This vicarious pulmonary development is eminently attainable in childhood, and therefore within our own gift; let it not be withheld. As even one day of this treatment is a gain, every succeeding day will be a further step towards a cure.

Is any further treatment necessary? Advanced and neglected cases may need a good deal more of it than could be discussed here. Early cases will certainly need very little. But for both sets of cases *inhalation* is of value. The simplest form is the *respiratory bib*, a twofold piece of flannel soaked in terebene or other inhalant, and enclosed in an open bag of jaconnette to be secured between the night-shirt and under-vest, just below the collar, so that the impregnated air is constantly being inhaled.

The dry bottle inhaler, filled with horsehair and with small pieces of blotting-paper steeped in the inhalant, is a variation on the same theme, acceptable to children for variety's sake, and because of the tube and its mouthpiece. Whether they blow or whether they suck through it, none of the strong fluid can come into their mouth.

These devices will generally render unnecessary the more searching treatment by the creasote chamber, even in cases of fœtor. But, in conclusion, they too may be superfluous if the rational treatment be conducted, instead of in a crowded town, in the pure air of the mountain or in the strong air of the sea. *Atmosphere and climate* are the most potent and sometimes the most indispensable of our adjuncts, and in children they are likely to produce results not to be equalled in the treatment of adults.

(*Read May the 8th,* 1908.)

Discussion.

Dr. Porter Parkinson said the Society was very much indebted to Dr. Ewart for his interesting and practical paper. He would like to hear the opinions of members on one particular mode of treatment, and, if they had tried it, their experience of it, namely, the treatment introduced many years ago by Dr. Vivian Poore, chiefly for the treatment of such conditions as fœtid bronchitis. He referred to the garlic treatment. One man came to the hospital about once a year, each time suffering from the same form of bronchitis—the fœtid variety. He came to Dr. Parkinson because he could get the garlic treatment, and that seemed to relieve all the symptoms in a remarkably short time. He gave a couple of grammes of garlic chopped up and put between two pieces of bread and butter. Of course the garlic was somewhat overpowering, but he knew it to be of value. In the treatment of bronchiectasis the case was somewhat different. The wall of the bronchiectatic cavity was not lined with normal mucous membrane, sometimes not by mucous membrane at all, therefore garlic treatment would not have the same value; still, he thought that in one or two cases of bronchiectasis some benefit had accrued from the garlic treatment. The garlic was excreted by the mucous membranes of the body, and apparently it had a rather powerful antiseptic action. Except for the garlic the diet of the man he had referred to was the ordinary full diet.

Dr. Harry Campbell said the point which occurred to him in regard to the apparatus exhibited by Dr. Ewart—of which he had no personal experience—was whether the child suffered in any way from the effort to keep the head erect. He thought there must be considerable strain on the posterior cervical muscles. He asked how long it was necessary or safe to keep a child in the apparatus.

Dr. G. L. Turnbull asked whether the patient was generally kept in the dorsal position during the night. He supposed the prone position would be impossible for so long.

Mr. Milner Burgess asked whether the treatment was graduated or sudden.

The Chairman (Dr. William Ewart) in reply, said he had been guilty of a good deal of garlic treatment. The odour was persistent and not easily forgotten or forgiven. No doubt it effected considerable benefit, but at the cost of considerable comfort in regard to other people. He had received a testimony to it from an outside patient in the neighbourhood of London, who received remarkable benefit from it; but he had suffered so much from the indirect results of such treatment that he did not now push it as much as formerly. Dr. Campbell's question as to the strain on the cervical muscles was a very important one. Though he had had many children on the apparatus he did not find any harm in that way. Indeed, it had an effect the opposite of that feared, for instead of overstraining, and thereby paralysing and weakening the cervical region, it gradually increased its power. Children who had been lying on their backs perpetually, with weak spines and back muscles, when put into the apparatus constantly tried to look about them, and there was thus a gradual strengthening of the muscles concerned. He was careful not to put them to that strain in extreme cases of rickety paralysis, but tried to work up towards strength by means of an elastic respiratory jacket. The dorsal position was the natural one for a patient to fall into at night, particularly the adult, and it was upon an adult he first tried the apparatus. Children would turn about between the restricting sandbags into almost any position, and the more they did so the better for them. The dorsal position was not of advantage for drainage, but the great point was the degree of inclination. The lungs were chiefly in the posterior part of the chest, and the bronchi occupied the posterior part of the lungs. The manner in which the treatment was begun was important. Quincke, who first started the prone position, approached it very carefully, and Jacobson, who wrote a long paper on it two years later, described how he was careful to accustom people to lie flat, and increase the slant more and more, as well as the periods of lying. An intermittent treatment was brought forward—an hour once or twice a day. With the adult he could start upon the treatment at once. He let the patient off it the first night, but on the second and third nights they would sleep with the bed well raised at the foot.

86. CASE FOR DIAGNOSIS.

By PHILIP G. LEE.

Nelly H—, a strong and healthy infant, aged 2 years and 6 months, was first seen by me a month ago; she had then marked wry-neck, no fever, appetite good, and was able to run about.

The following week she was brought to me with extreme retraction of the neck. On her way to my house she fell on the road, and when I saw her I found both legs paralysed. I then admitted her into the hospital. Four days after admission she had three convulsions, and the retraction of the neck disappeared completely.

Her condition (May the 2nd) as to general health is excellent, save for the paralysis. Her legs are wasting with the reaction of degeneration well marked. To assume the sitting position she has to be propped up in bed. The bladder and rectum are unaffected. The peculiar features of the case to my mind are the first onset, suggesting meningeal involvement, followed by symptoms of polio-myelitis, along with entire absence of fever or even malaise.

I would like to have the opinion of the members on this case as I have not met with one like it.

[In the absence of Mr. Philip G. Lee his notes were read by the Secretary.] (*Read May the 8th*, 1908.)

Discussion.

Dr. F. Parkes Weber said the case seemed from the description to be one of acute anterior poliomyelitis, beginning with retraction of the neck, and it would not be the only case of the kind which had begun in that way. Possibly slight posterior basal meningitis was present at the commencement in some cases of infantile paralysis.* In a typical case of acute anterior poliomyelitis with paralysis of the right leg in a child under his care at the German Hospital, there was temporary well-marked cervical opisthotonos at an early stage of the disease. In his case the retraction of the neck apparently was first noticed two or three days after the weakness in the leg.

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* Compare "Poliomyelitis Acuta und Meningitis Cerebrospinalis," by Dr. E. Tiedmann, in the 'Münch. Med. Woch.,' October the 23rd, 1906, p. 2095.

Dr. Porter Parkinson asked Dr. Weber whether he considered that an acute anterior poliomyelitis was likely to occur in the sudden way which that case apparently did. He understood the patient was on the way to the doctor's when the symptoms suddenly appeared. There seemed only one explanation of such a sudden onset, namely a vascular lesion; there might have been such a lesion superadded to poliomyelitis.

The Chairman (Dr. William Ewart) expressed the thanks of the Society for the contribution. Possibly there was much more which might have been said about it, but it was probably acute anterior poliomyelitis with an unusual commencement. If the child had been kept in bed he did not think it would have revealed such a degree of acuteness as it did.

87. A CASE OF AORTIC AND MITRAL STENOSIS.

By O. K. WILLIAMSON, M.D.

(INTRODUCED.)

A. A—, a boy, aged 8 years, came up to the out-patient department on February the 20th, 1908, complaining of pain in the left side of the chest, associated with occasional faintness and palpitation.

There was a history of pneumonia three years before this time, but none of rheumatism or tonsillitis. One sister has had chorea.

The child has a good colour. There is no clubbing of fingers. On examination the heart's apex beat is found to be in the fifth left intercostal space $\frac{1}{2}$ inch outside the nipple line. The impulse is waving in character. There is a presystolic thrill after exertion. There is no increase of cardiac dulness to the right, but there is slight impairment of resonance in the second left interspace. At the apex both sounds are audible, the first being short. At and within the apex can be heard a crescendo presystolic murmur, also a systolic murmur which is not conducted into the axilla. At the second right interspace both sounds are audible, also a harsh systolic murmur having its maximum intensity at this spot, nearly replacing the first sound and being conducted up to the clavicle and into the carotids. There is no diastolic murmur. At the second left interspace the systolic murmur is audible and the second sound is markedly accentuated. The pulse is small and regular, the vessel is full between the beats.

The arterial blood-pressure on April the 10th was found to be—systolic 91 mm., diastolic (approximately) 70 mm. The urine contained no albumin. Slight choreiform movements were observed. The condition has continued practically unchanged up till the present time.

(*Exhibited May the 8th*, 1908,)

Discussion.

Dr. Porter Parkinson asked whether Dr. Williamson considered it was a congenital or acquired heart lesion.

Dr. T. R. Whipham asked what was the result of the count of the red blood-corpuscles. If there was polycythæmia it might point to the condition being congenital rather than rheumatic. Personally, however, he thought that the lesions were acquired.

Dr. O. K. Williamson (introduced), in reply, said the symptoms were not severe, and that the child was able to walk about without distress. He thought that most likely it was rheumatic in nature, although in the absence of a rheumatic history there was no proof of that. The child had a certain amount of chorea, therefore he thought it was very likely an acquired condition. He had not had the blood examined. There was no symptom pointing to a congenital lesion. The symptoms did not date back more than two years.

88. AN INFANT WITH SUPERNUMERARY DIGITS ON HANDS AND FEET.

By HUGH LETT, Ch.B.

The baby, aged 2 months, was a girl, and the supernumerary digit on the right hand was attached at the level of the head of the fifth metacarpal bone. Its attachment was fairly firm, and the digit contained two short phalanges. The digit on the left hand was attached by a very narrow pedicle in a corresponding position, and also contained two phalanges. There were no additional metacarpal bones in either hand. The metacarpal bones were normal.

The extra digits on the feet were firmly attached, and in a line with the other digits; there were no additional nor any abnormal metatarsals. The mother of the child showed accessory digits on her own hands and feet. Those on the hands were firmly attached, and about the thickness of her little finger. She was able to flex and extend them slightly.

She had had five children, and brought a son of ten who also had accessory digits on hands and feet. The other children were normal. The cases were brought forward more particularly to show how useful these accessory digits were, if they had a firm attachment, to members of the artisan class. The mother found her extra digits most useful, and would not have them removed on any account. (*Exhibited May the 8th*, 1908.)

89. A GIRL AGED TEN YEARS WITH FORWARD DISLOCATION OF THE INNER END OF THE CLAVICLE.

By HUGH LETT, Ch.B.

She had fallen on to her shoulder five weeks previously, and the inner end of the clavicle was displaced forwards. Attention was drawn to the great rarity of the condition in a child. (*Exhibited May the 8th*, 1908.)

90. A CASE OF MULTIPLE ENCHONDROSES.

By HUGH LETT, Ch.B.

The case, a boy, aged 9 years, was remarkable for the extraordinary number of the enchondroses. They were not only present in large numbers on the long bones, but could also be felt on the scapulæ, ilia and skull. At present they caused no disability. He showed many signs of rickets. (*Exhibited May the 8th*, 1908.)

91. A CHILD OF FIFTEEN MONTHS WITH HYDROCEPHALUS, GENU RECURVATUM, AND TALIPES VALGUS.

By HUGH LETT, Ch.B.

The infant had been brought to the Belgrave Hospital nine months previously, and was shown to the Society as an example of the beneficial effects which could be produced on genu recurvatum by persistent massage and passive movements. When she was first seen the legs could not even be brought into a straight line with the thigh, but the left knee could now be flexed through 45 degrees, and the right could be just slightly fixed.

(*Exhibited May the 8th*, 1908.)

92. AN ABDOMINAL TUMOUR.

By J. PORTER PARKINSON, M.D.

A girl, aged 1 year and 10 months, was admitted into hospital for vomiting and enlargement of the abdomen. She had had no previous illnesses, and there were no abnormal signs in the chest. The abdomen was somewhat distended, the spleen was just palpable, and below it on the left side of the abdomen was a lobulated mass, firm in consistence, movable, and about the size of a Tangerine orange. The lump could also be felt *per rectum*, and felt like a mass of enlarged glands. He considered it to be a mass of tuberculous glands, and asked the opinion of the Society as to whether removal was advisable. There was no family history of tuberculosis obtainable.

(*Exhibited May the 8th*, 1908.)

Discussion.

Mr. Hugh Lett said the surgical treatment of tuberculous glands in the abdomen had not advanced far. A few cases had been operated upon and the glands successfully removed. There was much to be said in favour of operations of the kind. If enlarged glands in the abdomen were tuberculous they formed a dangerous focus, first, because it might spread the disease to other parts, and secondly, in that it might be a cause of tuberculous peritonitis, as well as adhesions to various parts of the intestines. In one case which he saw, tuberculous glands came down to the appendix; that formed a band, under which subsequently a loop of intestine passed, and it was strangulated. Meckel's diverticulum had become loose in the abdomen. If there was a movable mass in the abdomen he thought it worth while to try operation. If tuberculous glands in the neck formed a large mass they should be removed, and the same was true of glands in the axilla; and in regard to the mediastinum, the surgeon had not yet advanced far enough to remove those, though he looked forward to the time when that would be possible.

Mr. Milner Burgess (Frinton-on-Sea) thought that if the mass was tubercular it should be removed. He recently had a case in which an abdominal mass was not removed, and the patient died of tubercle in the lung.

The Chairman (Dr. William Ewart) said he had but little practical experience of his own on the matter, but if the surgeon could help them it would be a decided gain in the matter of safety to have such glands removed at the earliest date.

Dr. Porter Parkinson, in reply, said he thought it was one of the cases in which surgery would be useful. There seemed to be no evidence of tubercle elsewhere, and the glands were freely movable. Arrangements had already been made with a surgical colleague to remove them.

93. CASES OF ACHONDROPLASIA.

By RUSSELL HOWARD, M.S.

Two cases of achondroplasia were exhibited which showed the usual deformities present in the disease. The scalp muscles had been dissected, and the exhibitor pointed out that their development was normal, the contrary being alleged by some writers.

(*Exhibited May the 8th*, 1908.)

94. AN ANENCEPHALIC FŒTUS.

By RUSSELL HOWARD, M.S.

(*Exhibited May the 8th*, 1908.)

95. FULL-TIME CHILD WITH MULTIPLE DEFORMITIES.

By RUSSELL HOWARD, M.S.

A FULL-TERM child was exhibited which showed the following deformities: Absence of the left arm, absence of the right arm below the elbow, absence of both lower extremities. There was no obvious cause for the condition, and the child died soon after birth.

(*Exhibited May the 8th*, 1908.)

96. THE WIGHTMAN LECTURE: ON THE DEFENSIVE ARRANGEMENTS OF THE BODY AS ILLUSTRATED BY THE INCIDENCE OF DISEASE IN CHILDREN AND ADULTS.

By SIR WATSON CHEYNE, Bart., C.B.

Gentlemen,—The choice of a subject for the present lecture has been a matter of very great difficulty, and if it has not been a very fortunate one I must crave your kind indulgence. It seems to me that the object of lectures such as these is best attained by making an attempt to present a wide view of some general subject and one suggestive of future research rather than to discuss some more limited matter which would be better suited for a paper at an ordinary meeting of the Society. Hence it was among the former class of topics that my choice lay, and it has occurred to me that the subject of the relative resisting power of children and adults against infective diseases was well worth special study.

In dealing with diseases of children and adults one is often struck by the fact that marked variations occur at different ages; indeed, it is the existence of these variations which justifies a partial separation of children's diseases from those of adults, and the establishment of separate children's hospitals, or, at any rate, of wards in general hospitals set apart for the treatment of children. In the present lecture I shall speak only of diseases which are due to the entrance of parasitic organisms from without with special reference to the points of difference in children and adults. I am not able to explain these differences satisfactorily, I can only indicate some of the points which arise in the hope that some members of this Society who have more time and more ability than I have will turn their attention to the study of these matters. I believe that the successful study of the differences in the resisting power of children to infective diseases as compared with adults will give the key to much of the obscurity and difficulty which still surround the questions of infection, protection, and immunity.

In looking into this subject we find that there are some diseases which practically only occur during childhood, and *vice versâ* that there are certain diseases common in adult life which hardly ever affect children. Again, we find that there are many infective diseases which attack both adults and children, but the young are more especially susceptible to some, while their elders are more susceptible to others. Or, again, we find that some diseases are more virulent in children than in adults, and *vice versâ.* Perhaps the most striking fact of all, and the one to which I wish especially to draw your attention, is that in diseases which are common to both children and adults the organs and tissues which are attacked by the virus may differ markedly according to the age of the patient.

That such differences in the incidence of disease in children and adults are very common is a point which hardly requires any elaboration. The *exanthemata,* for instance, are very often spoken of as children's ailments, and some explain the fact that they are most frequent in children by saying that practically all individuals are highly susceptible to them, and being widely spread, no individual can live long in the world without being exposed to infection. This may be in part true, but I can hardly accept it as a complete explanation of the facts ; I cannot but think that, quite irrespective of opportunities of infection, the susceptibility of the individual varies according to age. Even in the case of children, the exanthemata differ among themselves as to the age when they most frequently occur : for example, measles is not uncommon during the first year of life, and something like 90 per cent. of the cases occur in children under five years of age, while scarlet fever rarely occurs in infants, although patients suffering from it may be present in the house or even in the same room, and the favourite age for scarlet fever is from three to ten years. Then, again, chickenpox occurs at a somewhat later period than scarlet fever; it is not so universally distributed, and it very rarely affects adults.

To take an organism whose life-history is known, we have a very good example of varying susceptibility at different ages in the case of the *pneumococcus*. This organism attacks the body more readily in childhood than in adult life, or as I would prefer to put it, the resisting power of the human body to the pneumococcus is apparently lower in children than in adults; thus it seems to be the fact that pneumococcal pneumonia is more common in children under five years of age than during any other quinquennial period. One of the most interesting points from a surgical point of view is the relation of the pneumococcus to pleurisy and empyema. The organisms which are most commonly at work in setting up these conditions are the pneumococcus, the *Streptococcus pyogenes*, and the tubercle bacillus; other organisms, such as *Bacillus coli*, staphylococcus, etc., are quite rare. Now, the relative frequency of these organisms in cases of empyema differs very markedly in children and adults. Thus, in children over 60 per cent. of the cases of empyema are due to the pneumococcus, about 15 per cent. to the streptococcus, and about 7 per cent. to the tubercle bacillus. In adults, on the other hand, the proportions are: Pneumococcus, 25 per cent.; streptococcus, 41 per cent.; and tubercle bacillus 18 per cent. Thus the pneumococcus is the most common cause of empyema in children and the streptococcus in adults. In children pneumonia is present in cases of pleurisy in from 25 to 50 per cent.; in adults it is present in only about 20 per cent. Further, pleurisy more often becomes purulent in children than in adults; indeed, empyema after pleurisy is about four or five times more common in the former than in the latter.

I think there can be little doubt that the meaning of these facts is that children have less resisting power against the invasion of the pneumococcus than is the case in adults, and this view is strengthened by the fact that we more often meet with pneumococcal invasion of other parts of the body than the lungs and pleura in children than in adults. Thus we find that while pneumococcal peritonitis occurs at all ages it is much more frequent in children, being most common between three and ten years of age. Similarly pneumococcal disease of joints is also most common in children, and the general invasion of the body by the pneumococcus is a very striking feature in early childhood and is most marked in infants, where we may have a regular pneumococcal septicaemia.

I need not give further examples of the general proposition that differences do occur in the incidence of various infective diseases in children and adults respectively, but may at once go on to the consideration of further points in connection with such differences. The question of infection is a very complex matter, and a variety of factors come into play in connection with it. On the one hand, we have the *invading parasites*, and on the other the *defensive arrangements* of the body against this invasion. I need not here go into the questions which relate to the parasites, their virulence, their habitat in the body, their habitat outside the body, the varying ease with which they gain access to the body, and so on, seeing that these points are the same whether the individual attacked is young or old. Passing, however, to the other partner in the infective process, namely, the animal body, we find that there are a variety of conditions which are necessary before the invading organisms can obtain a foothold in the body.

The *first line of defence* of the body resides in all probability in the epithelial tissues which oppose the entrance of infective agents into the body, and differences in the amount of resistance which they oppose to the entrance of organisms exist at different periods of life.

It is not as yet possible to define the exact meaning of the condition of the epithelium which enables it to oppose invasion. It clearly must be a local power residing in the epithelial cells themselves, or in the fixed cells on which they are situated, rather than any general condition such as the presence of opsonins in the blood, etc. It is interesting to note, as shown long ago by Lister, that where the epithelium lining a canal, the sides of which are normally in contact, is intact and healthy, bacteria cannot spread along that canal. This is true in the case of many ducts, such as the urethra, the mammary ducts, the salivary ducts, etc., and Lister used to point to this as a proof of the vital power of the tissues in preventing the growth of bacteria.

A very good example of variation in the defensive power of epithelium according to age is furnished by the behaviour of the *gonococcus,* and two points occur in connection with this organism in children. In the first place there are reasons for believing that the susceptibility of children to the invasion of the gonococcus is greater than in adults; and in the second place the organism attacks epithelial tissues in children, which are more or less immune in adults. The very mode in which infection occurs in the two cases seems to imply a diminished resisting power on the part of children, for in them infection is usually accidental, being conveyed by dirty sponges, soiled linen, etc. No doubt the anatomical development of the parts such as the vulva, as age advances, may render accidental infection less easy in adults than in children, but still it occurs more readily in the latter than this would account for. The incubation period in children is also apparently shorter than in adults. The disease as it occurs in young people after infection is usually more severe than in adults, and there are various observations which seem to show that discharges which are no longer capable of infecting the mucous membranes in adults are virulent for those of children. The greater readiness with which the conjunctiva becomes infected in children may also point to increased susceptibility to the gonococcus, though possibly the fact that in infants the conjunctiva is only one layer of cells thick may have something to do with the greater susceptibility. The special interest, however, centres in the location of the disease in children and adults respectively—namely, in the fact that the vaginal mucous membrane is more often and more violently attacked by the gonococcus than in adults. In adults, as a matter of fact, the disease is usually located in the cervix uteri or in the mucous membrane of the urethra, while involvement of the vaginal mucous membrane is relatively rare and slight. Some, indeed, altogether deny the susceptibility of the vaginal mucous membrane to the gonococcus in adults, and hold that when it is diseased it is only affected by a sort of desquamative catarrh caused by the maceration of the mucous membrane from the passage of the pus over it. While the vaginal mucous membrane in children is thus highly susceptible to the gonococcus it is curious that other complications caused by this organism are not so common nor so severe in children as in adults. No doubt gonococcal peritonitis does occur, and it is quite likely that the so-called spontaneous peritonitis in young girls is often of gonococcal origin. Gonococcal cystitis is also not uncommon, but gonococcal infection of joints is rarer than in adults. It most commonly affects only one joint, usually the knee; hydrarthrosis is not observed, ankylosis is very rare, and on the whole the course of gonococcal joint troubles is more benign and not so chronic as in adults.

Another example of the relation of organisms to the first line of defence is furnished by the behaviour of the pyogenic organisms in the skin. *Multiple abscesses of the skin* and subcutaneous tissues are quite common in infants. Impetigo is also common, but boils rarely occur. The reverse is the case in adults, where multiple abscesses of the skin are quite uncommon, their place being taken by boils. What the explanation of the difference is is not quite clear, but it is quite possible that in this instance it is in the main a question of differences in the anatomical structure of the skin rather than of general defensive arrangements. Thus in children the desquamation of the skin is more active and there is also incomplete development of the corneous layer of the epidermis in various places; in fact, the epidermis in children is very thin and extremely fragile and it desquamates readily, leaving the rete mucosum exposed. The papillary layer of the skin is also very rich in blood-vessels and reacts readily to all irritations.

Having overcome the first line of defence, the parasites either produce a local disease at that part, as we have seen in the case of the gonococcus, or else (and this happens in the majority of cases) they pass on into the lymph or blood stream and are carried to various parts of the body. In this latter case, having passed the epithelial line of defence, they then find themselves in closed tubes lined with endothelium. Apparently some forms of bacteria, having reached the circulating blood, can multiply in it and set up disease, but the great majority, and probably to a greater or less extent all, have to find a local resting place; if they fail to do so they quickly die out. We may imagine the bacteria being carried along in the circulation through the various tissues of the body, investigating these tissues as they pass through them till at last they find somewhere or other in the endothelial lining of the vessels conditions which are favourable for their growth or penetration, and therefore we may assume the existence of a *second defensive arrangement—namely, the endothelium.* Having found a weak spot in the endothelial lining they grow there and produce disease or else they pass through the wall of the vessel as they did through the epithelium, and multiply in the tissues outside. We have evidence that in a good many cases the parasites grow in the endothelial lining of the vessels. In syphilis, for example, endarteritis is the chief lesion and is probably due to localisation of the spirochæta in the inner coat of the small vessels. In the case of tubercle also I pointed out some years ago in my essay for the Astley Cooper prize that many tubercles are formed in the first instance in the interior of the blood-vessels possibly by proliferation of the endothelium, and in some specimens one can see tubercles in all stages clearly illustrating this point.

What the local predisposition on the part of the endothelium and the tissues signifies it is difficult to say. To some slight extent it may have to do with the *general trophic condition of the parts,* as influenced by the action of the nerves. In connection with this point I may refer to the interesting observations which have been made by Mr. Cheatle on the sites of development of epithelial cancer and its mode of spread, in which he produces some very remarkable examples of the spread of cancer along certain definite nerve areas, avoiding neighbouring areas supplied by other nerves. To a considerable extent it may be a question of *pabulum.* That the nature of the pabulum is of very great importance in the artificial cultivation of micro-organisms is, of course, well known. We know, for example, that it is not easy to cultivate the tubercle bacillus outside the body; we know that in cultivating the gonococcus it is necessary to have a little human blood on the surface of the cultivating medium to enable the growth to start; and we know that up to the present investigators have not been able to find a soil and external conditions which are suitable for the growth of various other infective organisms, such as those of leprosy, etc. It is, therefore, not at all unlikely that in the living body the question of suitable pabulum may have a good deal to do with the localisation and growth of the organisms.

An example which seems to point in this direction is that of *ringworm of the scalp.* This disease may affect children of all ages but is rarely noticed before three or after fourteen years of age; it never appears after the age of fifteen, and adults may mix freely with children suffering with ringworm of the scalp without being attacked by the disease, while on the other hand it is extremely infectious for children and will run through a school very quickly. The disease if left to itself disappears spontaneously at puberty, fresh and healthy hairs growing on the affected site. This is a very curious case and it seems difficult to frame a reasonable explanation. The fungus penetrates into the hairs close to the roots, but it evidently does not destroy the hair bulbs, otherwise the hairs would not grow again. It is therefore growing in what is practically dead tissue, or at the best in tissue of extremely low vitality, and the curious thing is that though it grows readily in this tissue in children it cannot grow in what is to all appearance the same material in persons over fifteen years of age. Is it that there is a difference in the chemical composition of the hairs in adults and children, and is it only in children that they provide suitable food for the fungus? Or is it that the fungus penetrates into tissue which still retains a certain amount of vitality, and there are more vitality and more resisting power in it in adults than in children? Whatever be the explanation, I think that this question of the suitability or unsuitability of the food which the parasite finds in the part which it invades must not be overlooked in considering the various factors which go to make up the defensive arrangements of the body.

Another example which may be mentioned in this connection is that of the common *threadworm*, which is only, or at any rate very much more often, found in children than in adults. Threadworms are very common in the large intestine of children, and they probably enter along with the food or drink. They occur in children at an age when their food is practically the same as adults, and yet threadworms are essentially a disease of childhood. May not this again be simply a question of pabulum? Is there a difference in the contents of the large intestine in children and adults, consisting in the presence of substances suitable for the existence of these worms in the former, such substances disappearing as the individual reaches adult life? Or is it that there is something in the intestinal secretions of adults which is inimical to the growth of the threadworm and which is absent in children? This might be an interesting physiological problem to work out. I can hardly think that in this case, at any rate, it can be a question of tissue resistance, for the worms grow in the fæces and can hardly be affected by the living cells lining the canal.

In this connection, also, I may refer to an interesting point—viz., that bacteria of the same species may acquire a special appetite, so to speak, for certain tissues. For example, while we look on the *Staphylococcus pyogenes* as an organism which may cause suppuration indifferently in any suitable tissue in which it may happen to find a proper resting place, we find that in certain cases where fresh developments of these organisms take place in the same individual, they are apt to occur in connection with similar tissues. Thus, I have repeatedly seen cases of staphylococcal osteo-myelitis in which fresh outbreaks of osteo-myelitis have occurred in several bones in succession without the occurrence of suppuration in other tissues of the body, as if the organisms which had settled in the bone in the first instance had acquired an appetite for bone, and when carried to other parts of the body again selected the growing ends of the bone for their development. This is perhaps still better seen in tubercle, where in cases of tuberculous disease of the bone we not uncommonly find that if fresh tuberculous disease occurs it is in other bones, and the tubercle may remain limited to bone for a long time. This is often the case, for example, in infants with tuberculous osteo-myelitis of the small bones of the hands and feet. Again, we have the tubercle bacilli in some cases showing a special tendency to grow in the lymphatic glands and not for a time spreading in other tissues, or in other cases growing in the skin and giving rise to lupus without leading to tuberculous disease elsewhere.

Stress has also been laid by some on the *anatomical arrangements* of the various parts as favouring the deposit of micro-organisms in certain situations. For example, in the case of osteo-myelitis the slower circulation at the epiphysial line may permit the deposit of organisms at that part, and thus account for the localisation in cases of this disease; indeed, it has been found experimentally that pigment granules injected into the blood are deposited in these situations. We cannot, however, accept this as the only, or indeed as a particularly important, factor, for many things point to something much more far-reaching than a mechanical deposition of bacteria. Thus the incidence of disease in various bones differs according to age and also according to sex. For example, osteo-myelitis is most common between the ages of twelve and eighteen, and at that age it is more frequent in the lower extremity than in the upper, and also more frequent in boys than in girls. On the other hand, the disease may attack children at a much earlier period of life, even below two years of age; in that case the upper extremity is affected nearly as often as the lower; the limitation of the disease in the first instance to the epiphysial line is not so marked, and, indeed, the lesions may occur in the epiphysis itself, and boys and girls are affected pretty equally. The anatomical arrangement of the part may possibly exercise some influence in favouring infection and determining the spread of the disease, but it is evidently a very minor factor.

It seems to me, then, that the localisation of the disease is in the main a vital question which may be summed up under the term "*local predisposition.*" As showing the complexity of this subject it may be pointed out that similar tissues in different parts of the body do not show the same susceptibility. Thus we know that the gonococcus seldom affects any mucous membranes except those of the urethra and the conjunctiva. And we shall presently see how tissues of various joints and bones in the body react differently to the tubercle bacillus at different ages. The same is the case if we take more general diseases. Thus in the case of plague Dr. W. J. Simpson points out that bubonic plague especially attacks the lymphatic glands in the groin and the axilla, especially in the groin, no matter whether the infection has occurred by punctures of the surface or in some less defined manner. We must therefore assume that the lymphatic glands in the groin differ in some way or other in their vital relations to these particular organisms from other lymphatic tissues in the body. To take another example, in acute tuberculosis, although we must assume that there is a general flooding of the blood stream with tubercle bacilli and that the circulating blood carries organisms all over the body, we nevertheless find that the bacilli are not deposited and grow anywhere indifferently, but that they select certain organs and tissues. For example, they are seldom found in the muscles and cellular tissues. In the case of infants who die from tuberculosis during the first four months of life it is found that the disease is practically always general, but the parts of the body in which the tubercles are deposited vary very considerably. Thus in post-mortem examinations of these infants tubercles are found in the lungs in all cases, in the bronchial glands in 99 per cent. of the cases, in the liver in 88 per cent., in the spleen in 86 per cent., in the brain in 24 per cent., and so on. And again, taking the brain for example, they are not equally deposited over the whole brain but prefer to locate themselves along the vessels at the base.

This local predisposition varies to a very marked extent with the age of the patient, the parts affected differing at different periods of life. Perhaps the best example of these variations is furnished by the incidence of *tuberculous diseases of bones and joints* at various ages. In the great majority of cases of tuberculous disease where the bacilli which escape into the blood stream are few in number a very close selection is made, and only one or at most a few local deposits occur, and that apparently only under the most favourable conditions. What these conditions are which favour the deposit of tubercle bacilli in a particular part we really do not know, but, as has just been said, they vary with age and to some extent with sex, and also with a variety of so-called predisposing causes, such as injury or chronic inflammation.

Some years ago I collected a large number of cases of diseases of bones and joints in adults and children which had been treated in hospital, and the following remarks are founded on these statistics. In the first instance, taking a general view of the relative frequency of disease in bones and joints at different ages, I would direct attention to the accompanying table, which gives the percentage frequency of the total cases of disease of bones and joints in my list in each quinquennial period up to the age of forty years.

TABLE I.—*Percentage Frequency of Cases of Disease of Bones and Joints in each Quinquennial Period up to the Age of forty years.*

Age-periods—years.	Percentage of total number of cases.	Age-periods—years.	Percentage of total number of cases.
1 to 5 . .	23·3	21 to 25 . .	8·5
6 ,, 10 . .	16·0	26 ,, 30 . .	8·8
11 ,, 15 . .	14·8	31 ,, 35 . .	4·0
16 ,, 20 . .	15·0	36 ,, 40 . .	3·0

This table indicates clearly the great importance of age in relation to the occurrence of these diseases, but it does not accurately represent the facts of the case. Thus it may quite well be that a greater number of cases occur during the first five years of life, because a greater number of persons are alive at that age than during any other quinquennial period. Conversely, for the same reason, it may turn out that the disease is much more frequent than appears to be the case during the later quinquennial periods, owing to the smaller number of persons alive at that time. Investigations of this kind have been made with regard to phthisis, and have led to the surprising result that in Copenhagen, Sweden, and various German cities with regard to which calculations have been made, the danger of phthisis in any given individual constantly increases with advancing age, and that in advanced life a larger proportion of the individuals alive at that age die from phthisis than at the period of life in which it has been supposed to be most common.

Employing the German statistics of the proportion of persons per 1000 alive at various ages, I have calculated the real frequency of the disease in my cases, and in the following table we have the ratio per 1000 of my cases of disease of the seven larger joints, beginning in each quinquennial period. In the second column I give the apparent frequency of the cases without relation to the number of persons alive at the different ages, and in the third column the real frequency as calculated by the above method. I only give the results up to forty-five years of age :

TABLE II.—*Ratio per* 1000 *of Cases of Disease of the Seven Larger Joints in Each Quinquennial Period up to the Age of Forty-five Years.*

Age-period—years.	Apparent frequency.	Real frequency.
1 to 5	232	167
6 ,, 10	153	134
11 ,, 15	150	145
16 ,, 20	153	164
21 ,, 25	85	98
26 ,, 30	88	120
31 ,, 35	41	60
36 ,, 40	30	48
41 ,, 45	20	42

From this table it will be seen broadly that the real frequency of these diseases is actually greater in advanced life than would appear from the ordinary statistics, but still a good deal less than in youth. In my list the disease commences most frequently between one and five years of age ; it then declines and rises again between fifteen and twenty years ; it subsequently steadily declines, with the exception of slight rises between thirty-five and forty years and forty-five and fifty years. The accuracy of the last numbers, which are not given here, is, however, very doubtful, because the cases in my list which commenced at those ages were quite few in number. Great differences also exist as to the period of life at which the disease commences in the different joints and bones, as is evident from the following table, in which I have indicated the percentage proportion of the cases of disease in each of the seven larger joints, commencing in each decade :

Table III.—*Percentage Proportion of Cases of Disease of each of the Seven Larger Joints in each of Five Decades.*

Joints.	Decades.				
	I.	II.	III.	IV.	V.
Hip	30·2	29·3	4·8	—	12·5
Knee	29·5	22·8	18·2	36·6	6·2
Ankle	5·4	5·9	3·6	3·3	12·5
Tarsus	4·6	5·9	8·4	3·3	18·7
Shoulder	—	1·6	4·8	—	—
Elbow	6·7	9·2	6·0	13·3	18·7
Wrist	0·6	8·4	15·8	13·3	6·2

Thus reading the table from above downwards we see that of the total cases of tuberculous bone and joint disease which commenced during the first decade, 30·2 per cent. were cases of hip-joint disease, 29·5 per cent. were cases of knee-joint disease, and so on. Reading the table from side to side, we see the frequency with which disease of each part commences in each decade ; thus hip-joint disease commences most often during the first decade, and its frequency diminishes very rapidly. The knee-joint is also very frequently attacked during the first decade, but the fall in its frequency is by no means so rapid, and so on.

Not only does the resisting power of the body vary in the same individual at different ages, but it is very interesting to note that it often varies very considerably in the two sexes. Thus, speaking of tuberculous diseases of bones and joints, I find that of the patients on my list 65 per cent. were males and only 35 per cent. were females. Indeed, most forms of surgical tuberculosis are more frequent in boys than in girls. The following table * gives the percentage relations of males and females (hospital in-patients) in regard to diseases of the seven larger joints, and it will be noticed that not only are males more frequently affected than females but that the proportion differs in different joints. And not only are there differences in the incidence of disease in the two sexes, but the disease in the female seems to be as a rule more benign than in the male. That is a very interesting matter, which, however, I have no time to discuss here. I may also remark that while this is the case with regard to diseases of joints it does not necessarily hold good with regard to some other forms of tuberculous disease. Thus tuberculous peritonitis is almost equally frequent in the two sexes, while lupus is much more common in females than in males.

TABLE IV.—*Percentage Relations of Males and Females in each Joint.*

	Hip.	Knee.	Ankle.	Tarsus.	Shoulder.	Elbow.	Wrist
Males .	59·7	57·6	81·9	85·8	50	74·3	75·9
Females	40·3	42·4	18·1	14·2	50	25·7	24·1

The predisposition and resisting power of the body, both local and general, to parasitic invasion vary very markedly under a variety of circumstances and are often *very sensitive to external conditions.* Even the *seasons of the year* apparently have their influence on the resisting power of the body. For example, erysipelas is more frequent at certain periods of the year than at others, especially during February and November, and lupus is said to improve in autumn and to become worse in spring.

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* I would not, however, press the actual percentages which are mentioned in this table, because in some of them, especially in the case of the shoulder, the number of patients was too small to be of value.

One of the most important causes which influence the local predisposition of a part is *injury*. In the case of the epithelial tissues injury is naturally of great importance in connection with their defensive action. If there is a solution of continuity in these tissues parasites may enter through a breach of the surface without being subjected to the action of the epithelium at all. On the other hand, without actually destroying the epithelium, the injury may so depress the vitality of the cells that they can no longer successfully resist the parasitic invasion.

A very good example of the effect of injury is furnished by the history of tuberculous disease of bones and joints, and the facts are of considerable interest. As a rule, where a case of joint disease is referred to an injury the history given is usually that of some slight injury, such as a strain or slight blow; indeed, it is noteworthy that after severe injuries in tuberculous subjects, such as fractures or amputations, tuberculous disease does not occur at the seat of injury. Thus, I can recall more than one instance of patients suffering from tuberculous joint disease who sustained fractures, in one case of the shaft of a bone, the epiphysis of which was already diseased, where the fractured ends have reunited without the development of any tuberculous disease at the seat of injury. And as regards amputation, unless the operation is carried out through parts which are actually diseased, one does not expect to have a tuberculous stump as a result, however many tuberculous lesions the patient may suffer from. We also not uncommonly notice that after tuberculous joint disease has become quiescent, or even apparently got well, it may recur as the result of a slight injury.

A further point which is worth mentioning is that injury to some joints seems more likely to be followed by tuberculous disease than injury to other joints. This fact is in accordance with the view which the above tables must lead us to assume, that the different joints vary in their predisposition to tuberculous disease, and that consequently injury is more likely to have an effect in determining its occurrence in those joints which are naturally the most predisposed.

Lastly, I may mention that it seems from my statistics, with which I need not trouble you here, as if injury were not only an active agent in the production of these diseases, but also determined a graver form. In my list the cases in which injury was given as a cause were on the whole more serious than those where no cause was assigned, whether we judge by the occurrence of suppuration, the results as regards complete recovery, or the severity of the treatment required for the cure.

One very obvious way in which injury may act in determining the occurrence and localisation of infective diseases is by leading to extravasation of blood, and if bacilli happen to be floating in the blood, to their consequent deposit in the part, in this way breaking down the second line of defence—namely, the endothelium—just as the first line of defence may be broken down. Apart from actual rupture of vessels and extravasation of blood, injury may favour the deposit of bacilli in the part, in that it leads to changes similar to those noticed in the early stage of inflammation. These changes involve, I believe, in the first instance, a diminution in the resisting power of the tissues. I fancy that at the present time such a view of the result of the early stage of inflammation would hardly be assented to, because the leucocytosis which accompanies it is looked on as a most important part of the protective arrangements of the body. I would, however, refer to Lister's early observations on inflammation, especially on the effects of inflammation on the activity of the pigment cells of the frog, to show what a paralysing effect irritation has in the first instance on the functions of the tissue cells of the affected part, and my belief is that the essential resisting power of a part as regards local infection lies in the tissue cells rather than in the adventitious leucocytes which come into it subsequently, and this may explain the greater severity of the disease after injury. I have formerly pointed out that where an extra large number of infective agents enters a part a more severe form of disease always results, and it must be remembered that this question of dosage of infective agents is, of course, relative to the resisting power of the part. Where, therefore, a similar quantity of bacteria has entered two parts, one of which has been rendered less resistant as the result of injury, this dose will act in the latter case in the same way as if a large dose had been administered under ordinary circumstances.

Resisting power is also influenced by a variety of conditions apart from those already mentioned, and these are often spoken of as *predisposing causes of disease.* There seems no reason to doubt that exposure to cold especially affecting a sensitive mucous membrane is a potent factor in permitting the entrance of pathogenic organisms, and very probably has a great deal to do with the penetration of such organisms as the pneumococcus into the body, especially into the lungs. *Bad hygiene, bad food, starvation, alcoholism,* etc., are also important agents in lowering the resisting power of the body to disease, and the same is frequently seen as the result of *other illnesses,* such as influenza, measles, chickenpox, etc. To take the case of multiple abscesses in the skin in childhood to which we have already referred, we find that they often follow acute and chronic illnesses. Thus, they are frequent during convalescence from infective diseases, such as typhoid fever, pneumonia, influenza, etc., and in the course of measles, scarlet fever, and smallpox abscesses and even gangrene of the skin frequently occur. They are also quite common in tuberculous children. Presumably, therefore, these diseases lower the resistance of the body to the staphylococcus, and yet, curiously enough, taking tuberculosis for example, I find no evidence that the opsonic index to staphylococcus is as a rule seriously lowered in cases of tuberculosis.

In connection with this question of variation in the resisting power at different ages a very interesting point arises which is worth mentioning here. One would naturally assume that if one individual is more susceptible than another to a disease he would also be affected by that disease in a more severe form, and no doubt this is true in a very considerable number of cases, but there are a number of very remarkable exceptions. In the case of the exanthemata, although I cannot speak with authority, I have seen it stated, and I have been brought up in the belief, that if an adult is attacked the disease is more likely to assume a severe type than in the case of a child. And yet we have already assumed that an adult is less susceptible than a child. In the case of pneumococcal invasion of the lungs, although the largest number of cases occur below twenty years of age and therefore presumably during the most susceptible period, the greatest mortality takes place in patients above twenty years of age. In the case of tubercle we find also that while it is most common in young people the disease is most obstinate and less amenable to treatment in old people. And again with regard to tubercle, while it is not common during the first year of life, when it does occur it assumes a very grave type owing chiefly to the fact that it is very apt to become generalised, and this is also true in the case of other infections, such as pneumococcal, streptococcal, etc. We have, however, many examples in the other direction. Typhus fever, for instance, is rarer in children than in adults, but it is not so dangerous in children as in adults. Again, typhoid fever is not so common in young children as in young adults, being most frequent between twenty and thirty years of age and quite exceptional below eight years of age. We may therefore assume that susceptibility to typhoid fever is small below eight years of age, that it reaches its height between twenty and thirty years of age, and again diminishes later. But the curious point is that the mortality increases as the age advances, being 11·3 per cent. in children below ten years of age and reaching 34 per cent. in persons over fifty years of age. Other examples of both these points might be given, but the above will suffice for our purpose.

A probable explanation of these facts is somewhat difficult to formulate, but the two following points are, I think, worth taking into consideration. In the first place, I have already referred to the question of relative dosage of bacteria in speaking of injury, and it is quite possible that this point also comes into play here. Thus, while a comparative small dose will suffice to set up the disease in a susceptible individual it requires a larger dose where the subject is less susceptible. And in accordance with the results which I obtained some years ago the resulting disease with the larger dose in the less susceptible individual would be more severe than that with the smaller dose in the more susceptible person. But in the second place I would point out that it is as a rule when the less susceptible individual is over the average age that the resulting disease is more severe, whereas this is not necessarily the case when he is under the average age, always excepting the first year of life. Hence it is probable that another factor besides resisting power comes into play in infective disease, and it is with the view of calling attention to this other possible factor that I have introduced the matter here; this factor may be indefinitely spoken of as *recuperative power*. It is quite probable that apart from the agents which actually take their place in the fight against the invading parasites there are other agencies which have to do with the maintenance of the health of the body and with the restoration of strength after it has been subjected to infection, and those I should group under the term "recuperative power." And I am inclined to think that some factor of this kind plays an important part in the recovery of patients from infective disease, quite apart from any anti-toxic or anti-bacterial agencies. Leaving out the question of infective diseases we know that as people get older they do not recover so quickly and perfectly from injury or illness as is the case in youth. To some extent, and probably to a considerable extent in old people, this has to do with the gradual onset of degenerative changes in the various tissues and perhaps more especially in connection with the blood-vessels. If I am correct in suspecting the existence of some further vital energy of this kind its importance in treatment cannot be overlooked, and especially it warns us not to pin our faith as regards treatment to one single point but to try to influence all the agencies which have to do with the well-being of the individual.

Time will not permit me to pursue this subject further. I could have mentioned many other facts in illustration of the points to which I have referred, but those which I have picked out seem sufficient to show the bearings of the matter and to indicate its great interest and importance. Especially, I think, there can be no doubt of the existence of local conditions in the tissues and organs of the body which have a very important bearing on the occurrence of infection and on recovery from it. Indeed, in my opinion, the local conditions, which we have grouped together under the heading of "local resisting power," are of much more importance than any general agency which may be present in the blood stream.

I have already indicated some points which have to be taken into consideration in connection with this question of local resisting power, but in spite of all the work that has been done we are still in the stage of theory. One point I may refer to in connection with this matter, and that is the remarkable difference as regards leucocytosis in the new-born and in later life, taking the example of erysipelas. Erysipelas shows very marked differences in gravity at various periods of life and under various circumstances, and it is more especially grave in the case of infants. Infantile erysipelas usually appears from the tenth to the fifteenth day after birth, and generally spreads from the umbilicus. Its mortality at that age is at least 50 per cent., but the mortality steadily falls in older children. On examining the parts in infantile erysipelas the interesting fact is observed that there is almost complete absence of local leucocytosis. In the adult, leucocytes group themselves in large numbers around and within the blood and lymphatic vessels, while in the new-born one finds the vessels dilated it is true, but this is due to the presence of masses of streptococci and not to leucocytes. Enlargement of the lymphatic glands, which also occurs in adults, may be very slight or absent in children.

On the other hand, leaving out the first year of life, there seems to be a slight increase in general leucocytosis in children under ten years of age as compared with older people in some of these infective conditions. This has been especially pointed out in cases of otitis and mastoiditis.

That the conditions summed up under the term "local resisting power" are much more important than the more general conditions seems to me to be certain, and I have mentioned quite a number of points which indicate this. I have especially referred to the varying local incidence of disease at different ages and under different conditions; and I may also mention another very striking point as showing the importance of local as compared with general defensive arrangements. It is this, that in some cases one finds a disease making good progress towards healing at one part of the body, while at the same time a fresh outbreak is taking place at another. Take, for example, the case of tertiary syphilitic ulcerations. One of the important diagnostic points after a tertiary ulcer has lasted for some time is the tendency which it has to heal at one side while it spreads at the other, in this way giving rise to the serpiginous form of these ulcers. If the healing were due to general conditions in the serum, why should it not occur all round, and why, if the resisting power has been so much improved as to lead to healing at one part, does the disease actually progress at another part in the vicinity. The same is frequently seen in the case of tuberculosis; it is not at all uncommon to find one tuberculous deposit healing or becoming quiescent, while another becomes active or a fresh one actually appears. A very good example of that is seen in tuberculosis of the epididymis, where the disease on the one side and also in the vesiculæ seminales may be steadily improving when all of a sudden the other side becomes affected and not uncommonly runs a more virulent and rapid course than the first. I have seen exactly the same thing happen on several occasions in cases of tuberculosis which had been under steady opsonin treatment for a long time, some of them over a year. Thus, in one case of tuberculous epididymitis which I recall the disease in the epididymis was particularly acute in the first instance, and I advised opsonic treatment rather than operation. Ultimately, however, as the testicle became completely destroyed by suppuration, castration became necessary. Opsonic treatment was still steadily continued under the best auspices, but in spite of that, residence at the seaside, etc., the disease appeared acutely in the other epididymis, while the treatment was still going on, and I had to perform an epididymectomy without delay in order to save the other testicle. The course which the disease followed was in reality the same as if no attempt had been made to increase the general resisting power.

At the present time it is the fashion to look on the opsonic content of the blood as the essential agent concerned in bringing about immunity and cure in the case of infective diseases. This is a matter on which neither time nor knowledge permits me to enter at length. I have, however, searched the literature, inquired among investigators, and had a few observations made for me without finding any marked differences in the opsonic index to different organisms at various periods of life corresponding to the variations in the incidence of disease, and in their course at different ages; nor has any difference in the opsonic index to various infective organisms been pointed out as regards sex, and yet, as we have seen, there are noticeable variations in the incidence and severity of these diseases in the two sexes. It must, however, be pointed out that this matter of alterations in the opsonic index in connection with age, sex, etc., has not yet been worked out from the point of view taken in this lecture, and we must not, therefore, lay too much stress on our present knowledge on this point. It is a matter which it would be quite worth while looking into. The only investigations actually bearing on this point which I have come across are some described by Dr. J. H. Wells in the May number of the 'Practitioner.' He has made observations on the opsonic index in infants under one year old, with the result that he finds marked differences in young babies as compared with older people. He comes to several conclusions, of which I may mention the following: "A low opsonic index is not diagnostic in children under one year old." "In infants a low opsonic index is not inconsistent with health and the child may be thriving well with a declining index." "The anti-bacterial defence in children cannot depend upon the opsonic content of the serum." These are very important conclusions, and if found to apply to a sufficiently large number of cases they raise the question whether the significance of the opsonic index and opsonins generally is being properly interpreted. Although infective troubles when they do occur in infants are, as a rule, more acute than in older children, as already instanced in the case of tuberculosis, erysipelas, etc., nevertheless infants do not develop these infections so readily as children do after the first or second year of life—in general terms their defensive power is greater. But if it is a fact that the anti-bacterial defence in infants does not depend on the opsonic content of the serum, and yet at that period their defensive powers are high, is it right to assume that at a later period of life the anti-bacterial defence does depend on the opsonic content of the serum? May there not be some other interpretation of the variations in the opsonic index than that which is put forward by Wright and which is being so extensively translated into practice?

I must confess that while there is much that is interesting and important in the extensive work which is at present being done on opsonins, it would be more convincing to me if the theories on this matter were less complete and simple and if the writings were tinged with a little philosophic doubt. Unfortunately, when we come to study the results of the application of these theories to practice they do not work out as they ought to. In some cases good apparently does result, while in others it is an open question whether the benefit which follows would not have occurred to an equal extent without the vaccination; in many instances, however, and my experience especially concerns tubercle, one cannot convince oneself that the slightest benefit has resulted—in some, indeed, the condition seems to have become worse. Nor even in the cases where benefit has resulted is it clear that the bacteria have been completely eradicated as one would expect if the theory were correct. I think that all we can say about these matters at present is that they are highly interesting from a scientific point of view and that their continued study is highly desirable, but that it is very doubtful if the opsonin theory is the complete solution of the problem. For my own part, I feel satisfied that something is still wanting, and I believe that the discovery of that something will be more likely to be made in connection with the study of the local conditions of resisting power than of the general conditions of immunity. The time has certainly not yet come when we can with advantage to our patients beat our knives and stethoscopes into hypodermic needles and syringes, discard all our past experience and knowledge, and transform ourselves into immunisators.

(*Delivered May the 22nd*, 1908.)

97. A CASE OF BAZIN'S DISEASE (ERYTHEMA INDURATUM).

By A DOUGLAS HEATH, M.D.

(Introduced.)

The patient was a boy, aged 9 years, small for his years, and of a somewhat frail build. The eruption had started two years ago on both legs. At the onset several purple-red spots were visible on the front and sides of the left leg and on the lower part of the right calf. Several similar areas also appeared on the front of the left thigh. All the skin lesions had cleared up at the time of showing the case, leaving white scars, except one large area on the lower part of the right calf. This was still of a dull red colour, thickened at the edges and depressed and scarred in the centre. An ulcer had followed the breaking down of the skin over this area which had run a very chronic course. The whole area measured two inches in width and one and a half inches in height.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

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98. A CASE OF TUBERCULOSIS OF THE SKIN.

By A. DOUGLAS HEATH, M.D.

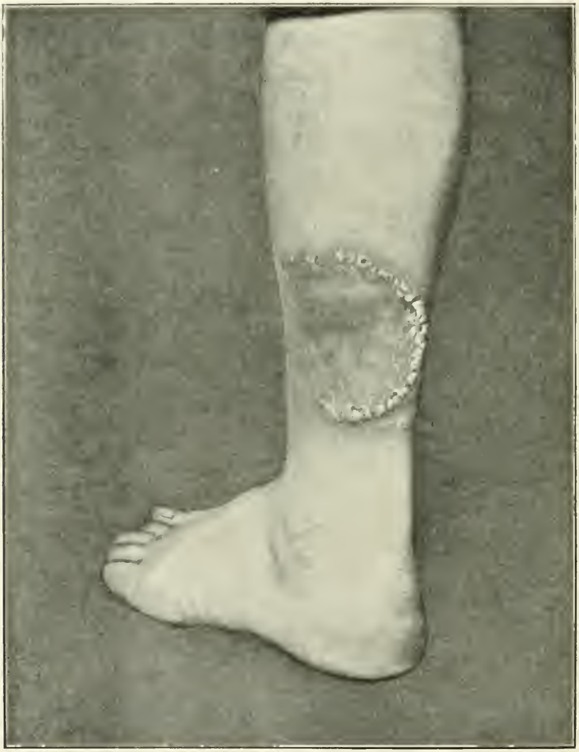
(Introduced.)

The patient was a boy, aged 8 years, of healthy appearance.

The eruption had appeared first three months previous to the patient being shown. Two large papule-like lesions had appeared on the outer side of the left arm, which rapidly broke down and suppurated, and a larger, although similar, focus of disease had appeared on the outer side of the left leg. The two smaller areas on the left arm rapidly healed up, leaving almost imperceptible scars. On the left leg, however, a small ulcer appeared, which also rapidly healed, but an infiltration of the skin around went on taking place until a large area of skin 3½ in. by 2½ in. was affected.

The appearance of this area is well shown in the photograph (Fig. 59). The margins of the area were very sharply defined and well raised above the level of the healthy skin. The colour of the margins was paler than of the central part of the diseased plaque, and the extending

FIG. 59.



margin had almost a glistening white appearance, and nowhere showed any areas of ulceration. The large dull-red central region of the patch was very distinctly indurated, but contained no nodules, and no scarring could be seen in it. The skin over this indurated area was quite smooth and showed scarcely any signs of desquamation.

A few small acneiform tuberculides of recent appearance on the extensor surface of the left arm could be seen at the time the patient was exhibited. The photograph shows very well the large size of the diseased area, and how the anterior rolled edge is beginning to break up and disappear whilst extension is still going on at the posterior margin.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

99. A CASE OF GRANULOMA PYOGENICUM.

By A. DOUGLAS HEATH, M.D.

(Introduced).

The patient was a girl, aged 12 years, who had a small wart on the scalp. This had been present, so her mother said, " from birth." About six months previous to the patient being shown, a red raspberry-like tumour had appeared on this wart. It could be seen to be growing from one half only of the wart, and was raised about ½ inch above its level. It bled profusely when touched, and discharged a thick yellow pus. Cultures yielded *Staphylococcus aureus.*

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

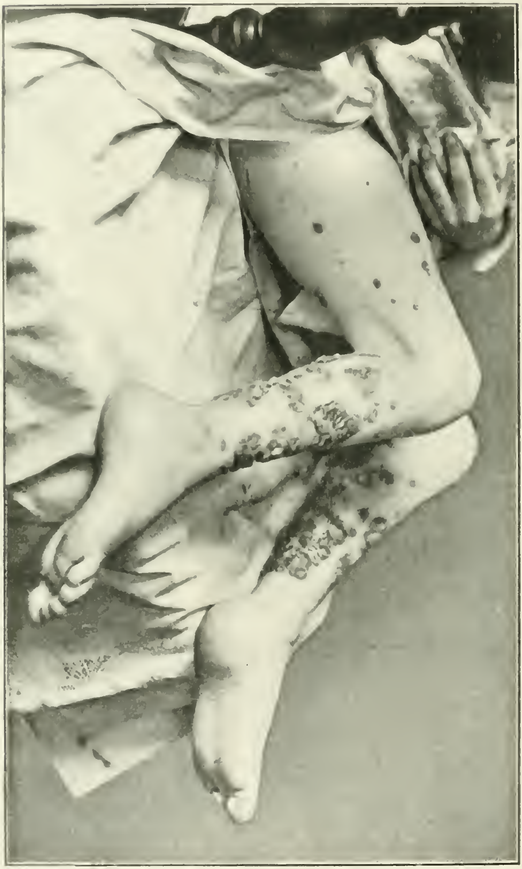
100. TWO CASES WITH BROMIDE ERUPTIONS.

By A. DOUGLAS HEATH, M.D.

(Introduced.)

Two children were exhibited showing granulomatous eruptions from the taking of bromides. Both cases had

Fig. 60.



occurred in the practice of the same doctor, and had been sent for diagnosis.

In the first patient, a child aged 6 years, medicine (? K. Br.) was given for what was thought to be whooping-cough, and the eruption soon afterwards developed. The photograph (Fig. 60) shows the large granulomatous eruption which was present when the child was first seen in December, 1907. At the time of showing, the patient's legs were covered with large cribriform scars left after the disappearance of the raised eruption.

In the second patient, a child aged 11 months, medicine had also been given for whooping-cough. The eruption was present on both sides of the face and on the legs. Granulomatous masses appeared exactly like those in the first patient. These tumour-like masses were covered with a thick white sodden cuticle underneath which granulation-like tissue could be seen. No typical pustules were present, and the granulomatous masses generally seemed to be quite dry and free from discharge. All cultures taken from them had been sterile.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

101. A CASE OF LUPUS.

By A. DOUGLAS HEATH, M.D.

(Introduced.)

A boy, aged 10 years, healthy-looking and strong, was exhibited suffering from a rare form of lupus.

On the back of the middle of the right thigh was a large horse-shoe-shaped patch of eruption 3½ inches long and 3 inches wide.

The horse-shoe-like band of affected skin was on an average about ¾ths inch in width and was much more raised at its extending margin than towards its centre. Running around almost the whole patch, and close to its extending raised margin, was a narrow shallow band of ulceration.

The large area of skin enclosed within this band of eruption was distinctly pigmented and showed slight scarring. Towards its periphery a few small nodules could be felt, which were of larger size in the band-like area itself.

Sections of skin removed from the growing border of the diseased area showed giant cells, epithelioid and small round cells and newly-formed fibrous tissue—in fact, a typically tubercular architecture. The ulceration could be seen to be quite superficial, and the prickle-cell layer was nowhere destroyed. There was a little acanthosis at the growing margins of the eruption.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

102. CONGENITAL SYPHILIS; CIRCINATE ERUPTION.

By A. DOUGLAS HEATH, M.D.

(INTRODUCED.)

THE patient was an infant, aged 9 months. He was the last of eight children, of whom seven are living and all are in good health. The mother denied ever having any eruptions of the skin, sores in the mouth or genitals, or any other signs of syphilis.

The father had been asked to attend for examination and had not done so, but his wife did not think he had ever had any eruptions. Seven children out of the eight had developed eruptions soon after birth.

The present circinate eruption, according to the mother, started when the child was 11 weeks of age, on the scalp. The eruption consisted of numerous circles and segments of circles freely scattered over the buttocks, thighs, legs and arms. In some regions, as on the flexor surface of the right arm, concentric areas of eruptions could be seen. These circinate syphilides were quite dry and were only very faintly scaly. They were of a faint pink colour, flat topped, and varied from $\frac{1}{8}$th to $\frac{1}{4}$th inch in width. The skin both within the rings and around them appeared quite healthy and unpigmented. Some of the larger rings of eruption had a diameter of 2 inches or more.

On each side of the anus there was a flat plaque of eruption with excoriation in the centre.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the* 20*th*, 1908.)

103. A PECULIAR DEFORMITY OF THE HANDS IN FOUR GENERATIONS.

By JAMES E. H. SAWYER, M.D.

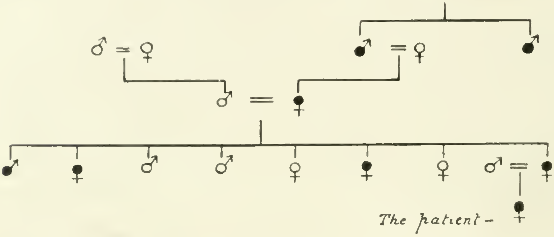
Girl, aged 7 years. Nothing abnormal was observed with the hands until she was about six months old. At that age it was first noticed that she could not quite close either fist or straighten any of the fingers to the normal extent. The girl had never had any pain in the hands, and the finger-joints had never been more swollen than they are at the present time.

On examination of the hands, all the phalangeal joints are slightly larger than normal, and the fingers cannot be fully extended nor flexed so as to touch the palms. The fingers do not deviate to the ulnar side, as occurs in rheumatoid arthritis. The thumbs appear quite normal, and their movements are not impaired. There are no other joints of the body affected, and the child seems quite healthy in every way.

The mother's hands are in a very similar condition to those of the child. They have always been the same as far as she can remember, and she has never had any pain in them. The mother has one brother and two sisters with the same deformity, and two brothers and two sisters not affected in this way. The mother's mother and her maternal grandfather and maternal great uncle are said to have had the same deformity.

The genealogical table shows which members of the family are affected. The condition is supposed to be the same in all of them, as none appear to have had any pain in their hands, but I have not had an opportunity to examine them for myself.

Fig. 61.



(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

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104. FOUR CASES OF CONGENITAL DISLOCATION OF THE HIP.

By FRANK BARNES, M.S.

(Introduced.)

Four cases of congenital dislocation of the hip were shown illustrative of the paper subsequently read before the Society. Two were cured, one being a case of double and the other a case of single dislocation. The third was in the process of cure, and the fourth was an incurable case.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

105. CASES OF UNRESOLVED PNEUMONIA.

By ARTHUR FOXWELL, M.D.

(Introduced.)

Case 1.—Irene D—, aged 5 years, admitted into the Queen's Hospital, Birmingham, April the 17th, 1907, with acute broncho-pneumonia. Family history good. Previous history of bronchial catarrhs. Discharged on May the 17th, 1907, with lungs not cleared.

Condition on June the 18th, 1908. About five or six times a week, *e. g.*, after running or excitement, she brings up several mouthfuls of "yellow phlegm." There is no expectoration nor cough at other times. General health fair. On coughing there are signs of dilated tubes at both posterior bases; there is also evidence of fibrosis. The heart is dilated and the liver enlarged.

Case 2.—Rosa N—, aged 17 years. On February the 20th, 1908, whilst suffering from a severe cold, she had tonsils and adenoids removed. She has never been well since. Admitted into the Queen's Hospital, Birmingham, on May the 20th with "chronic pleuro-pneumonia of the base of the right lung," with a slighter condition at the left base and an enlarged spleen. The sputum was very fœtid, contained some Gram-positive diplococci unlike pneumococci, but no tubercle bacilli were found. Temperature 99° to 102° F., irregular.

At first the signs of dilated tubes were evident, but now these are obscure; the sputum has fallen from five ounces to two drachms, the fœtor has gone, as well as her septic pallor. The treatment has been thorough emptying of chest twice a day, with the inhalation of a 5 per cent. solution of formalin. The signs of fibrosis of lung and pleura still exist.

Case 3.—John G—, aged 13 years. Six years ago adenoids and tonsils were removed during an acute catarrh. Severe double pneumonia followed, ending in double-bronchiectasis with fœtid sputum. By emptying chest twice daily the fœtor went, but the bronchiectasis with purulent sputum still exists. The boy himself seems well, plays and works well at school, but the breath is short and the fingers clubbed.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th*, 1908.)

106. EFFECT OF SCHOTT MOVEMENTS IN HEART-DISEASE.

By ARTHUR FOXWELL, M.D.

(Introduced.)

Case 1.—William T—, aged 21 years. Failing compensation in old severe aortic and mitral regurgitation. After a month in bed the pulse was reduced to 80. He was then allowed to sit up for one and a half hours daily for a week. His usual pulse-rate was then raised to 100, and on the average it was thirteen beats quicker after sitting up than before. He was again kept in bed and Schott movements (arms only for fifteen to thirty minutes daily) were prescribed and continued for three weeks. His general pulse-rate fell to 90, and on the average it was twelve beats slower half an hour after the movements than before. He was then allowed to sit up for an hour, the movements being continued; the general pulse-rate remained about 90, the slowing after the movements remained about twelve, and the rate after sitting up was also, on the average, eight beats slower.

Case 2.—Philip Hunt, aged 11 years. Mitral disease after recent rheumatic fever. Here Schott movements for fifteen minutes produced a fall in the pulse-rate of fifteen (average of a week). He then sat up one hour, and the movements continued to produce the same fall of fifteen, and there was a slight fall of four after being up. He next had the movements *whilst* he was up; now the fall in pulse-rate after was reduced to ten and there was no fall obtained by sitting up, that is, the two exertions taken simultaneously were as much as the heart could stand. Dr. Foxwell is glad to record these two cases, for in the large majority of hospital patients in whom he has tried Schott movements the result has been negative or injurious.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th, 1908.*)

107. SPLENIC ANÆMIA—BANTI'S VARIETY.

By ARTHUR FOXWELL, M.D.

(INTRODUCED.)

BERTRAM —, aged 15¼ years. Family history good. Patient was quite well till five years of age, when jaundice was first noticed; this has continued almost without change ever since, and he has never been in good health, but has attended a hospital as an out-patient except when confined to bed in hospital or at home.

At seven anæmia was first noticed.

He was first admitted into the Queen's Hospital, Birmingham, in March, 1905, with severe epistaxis and slight pyrexia. The liver was then one inch below the ribs and the spleen reached to the umbilicus. Neither was unduly hard and neither has varied much in size since then. The epistaxis has continued on the average about twice a week, usually in moderate quantity. No enlargement of lymph glands has ever been noted.

Four blood counts made in March and April, 1905, gave this result:

Red corpuscles, 3,660,000 per c.mm.—much poikilocytosis, but no megalo- or microcytes, nor nucleated; white corpuscles, 10,000 per c.mm.; hæmoglobin, 54 per cent.

June, 1908, the blood count was: Red corpuscles, 3,062,500 per c.mm.; white corpuscles, 10,000 per c.mm.; hæmoglobin, 95 per cent. White differential count gave: Polymorphonuclears, 67 per cent.; small lymphocytes, 24 per cent.; large lymphocytes, 7 per cent.; myelocytes, 1 per cent.; eosinophiles, 1 per cent.; basophiles, 0·6 per cent. The red cells were normal in character.

There is considerable arterio-sclerosis. The average of three examinations of the urine gave specific gravity 1010, no albumin, no sugar, urea 0·8 per cent.; nothing abnormal seen by microscope; quantity in twenty-four hours 24 oz. The boy is now intelligent, pale, jaundiced slightly, in fair but delicate health, able to work as a jeweller except during the epistaxis.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th*, 1908.)

108. TWO CASES OF PSATHYROSIS.

By FRANK BARNES, M.S.

(Introduced.)

Case I.—A boy, aged 8 years, suffering from osteopsathyrosis.

He has never walked, and since his birth has had no less than eighteen separate fractures of the thighs and arms. A radiograph shows the site of some of these in the femora, which also displays a marked condition of coxa vara. The pelvis is of a marked tri-radiate character and the chest wall exhibits a flattening from side to side and a prominent sternum, giving it a barrel-shaped appearance sometimes seen in rickets. The boy's sister, aged ten years (Case 2), also suffers from the same condition, having had a similar number of fractures.

The children are the middle two of a family of six, the first two and the last being perfectly healthy.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

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109. HEREDITARY ATROPHY OF THE OPTIC NERVES.

BY J. JAMESON EVANS, M.D.

(INTRODUCED.)

A BOY, aged 15 years, suffering from hereditary atrophy of the optic nerves. Two uncles of the patient were similarly afflicted, but the disease could not be traced farther back in the family. The patient's first symptoms appeared five years ago. He showed no evidence of any nervous or other general disease. His sight is barely $\frac{6}{60}$ in each eye, there being central scotomata in each eye. Peripheral fields are full. The disease has not varied to any appreciable extent during the last three years.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

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110. CASES OF HEREDITARY NYSTAGMUS.

BY J. JAMESON EVANS, M.D.

(INTRODUCED.)

A BOY, aged 10 years, and a baby, aged 2 years, uncle and nephew respectively, suffering from hereditary nystagmus. The affliction can be traced through four generations. It was always transmitted through the females, whom it never affected. The nystagmus is of a

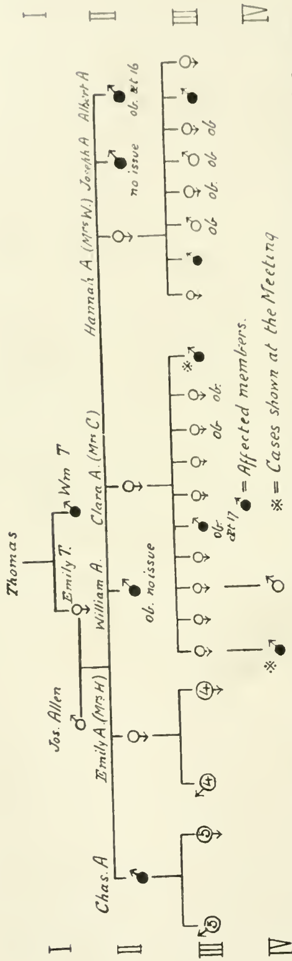


FIG. 62.—Genealogical tree illustrating Dr. Jameson Evans' Cases of Hereditary Nystagmus. Large circles with figures enclosed indicate collective number of children of each sex. Those marked ob. died at an early age except where marked.

slow lateral type with a slight amount of rotation. There is a considerable amount of ametropia, generally hypermetropic astigmatism, associated with it, and the vision in all the affected members is markedly reduced. All the affected cases had very light hair in infancy, but this became darker as age advanced. The irides are grey, but in no case is there definite albinism. The nystagmus in the case of the baby is not so marked as it was twelve months ago.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

III. GLIOMA AND PSEUDO-GLIOMA.

By J. JAMESON EVANS, M.D.

(INTRODUCED.)

GROSS sections of eyes removed for glioma and pseudo-glioma, and microscopic sections of the glioma were shown.

The latter displayed the usual small, round-celled growth proceeding from the retina and secondarily affecting the optic nerve and choroid. Deposits could also be seen in the suspensory ligament, lens capsule, and iris.

The pseudo-glioma had been removed from a boy, aged 3 years, whose father and mother were tuberculous. The child's other eye showed extensive pearly-white degenerative spots in the choroid at the posterior pole.

The excised eye showed a complete detachment of the retina. The subretinal fluid was albuminuous and contained cholesterin, and was very turbid in the neighbourhood of the choroid. The latter was completely atrophied anteriorly. The exact nature of the disease had not been decided.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

112. A CASE OF CONGENITAL ANIRIDIA.

By J. JAMESON EVANS, M.D.

(Introduced.)

A female child, aged 2 years, suffering from congenital aniridia in each eye. There is no history of a previous case of a similar nature in the family. A very narrow rim of iris can be seen in some parts of the circumference of the pupils, but the edge of the lens and the suspensory ligament are everywhere visible. In the left eye there is a very small anterior polar cataract and a more extensive posterior polar cataract.

The eyes are otherwise healthy.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

113. CASE OF ANTERIOR CURVATURE OF THE TIBIA.

By ALBERT LUCAS.

(Introduced.)

A boy, aged 13 years. There is an extensive anterior curvature of the left tibia. The curvature has developed in the last six months. It is regarded as due to congenital syphilis, although there is no history or other evidence of that affection.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

114. NINE CASES OF INTUSSUSCEPTION WITH RECURRENCE IN ONE CASE.

By F. VICTOR MILWARD.

(Introduced.)

In the nine cases of intussusception tabulated herewith, seven occurred in boys and two in girls ; six were under one year of age and three were over one year. In three cases the intussusception had lasted over twenty-four hours, but in No. 7, in which the duration was stated to have been seventy-two hours, and the attack a sudden one, whether an intussusception was the commencement or not of the illness is uncertain. Omitting No. 2, the recurrent case, there were six ileo-cæcal intussusceptions and three of the ileo-colic variety. Of the latter, two recovered and one died of injury to the intestine caused by the efforts at reduction. Case No. 1, which recurred three times and which was operated on by the reporter the first and last time, was found post-mortem to have a thickening of the submucous tissue in the terminal inch of the ileum. The overgrowth was of a lymphoid nature, producing a papillomatous appearance when viewed from inside, and causing a distinct narrowing of the bowel.

No.	Sex.	Age.	Date of operation.	Duration of symptoms	Variety.	Length of stay in hospital.	Result.	Remarks.
1	Male	$\frac{6}{12}$	Oct. 4, 1904	30 hrs.	Ileo-cæcal	16 days	C.	—
2*	,,	$1\frac{6}{12}$	Sept. 25, 1905	6 hrs.	,,	1 day	D.	At the time of the third operation the child was 18 months old.
3	Male	$\frac{7}{12}$	June 5, 1905	5 hrs.	Ileo-colic	21 days	C.	Died 2 months later of marasmus.
4	Female	$\frac{6}{12}$	June 16, 1905	48 hrs.	,,	12 hrs.	D.	Great difficulty in reduction.
5	,,	$\frac{9}{12}$	Aug. 3, 1905	12 hrs.	Ileo-cæcal	11 days	C.	—
6	Male	$\frac{8}{12}$	Nov. 26, 1906	—	,,	16 days	C.	Died 3 months later; cause unknown.
7	,,	$\frac{7}{12}$	May 16, 1907	72 hrs.	,,	21 days	C.	—
8	,,	2	April 20, 1908	14 hrs.	Ileo-colic, becoming ileo-cæcal	21 days	C.	—
9	,,	2	May 19, 1908	3 hrs.	Ileo-cæcal	20 days	C.	—
10	,,	4	May 18, 1908	11 hrs.	,,	22 days	C.	—

Patients, 9; number of operations, 10; operations successful, 8; cases died, 2.

2* Same as No. 1. A second operation was performed by Mr. Gamgee, August the 7th, 1905; result, C. The intussusception again recurred, and a third operation was performed by Mr. Milward, September the 25th, 1905; result, D.

Had the child recovered from the third operation, it was determined to short-circuit or excise the ileo-cæcal region in order to prevent a further recurrence. The child died of shock.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

115. A CASE OF DEPRESSED FRACTURE OF THE RIGHT PARIETAL REGION WITH LEFT HEMIPLEGIA IN A BOY, AGED SIX YEARS.

By F. VICTOR MILWARD.

(Introduced.)

This boy was admitted into the General Hospital, Birmingham, in April, 1908. Eleven days before admission a stone weighing 80 lbs. had fallen on his head, and since that time his condition had been considered too hopeless to be benefited by surgical treatment.

On admission.—He had a suppurating wound of the right parietal region with paresis of the left side of the face, the arm, and slightly also of the left leg. He was semi-conscious and very irritable and restless, and for the most part lay curled up in bed, resenting any interference. Temperature 99·4°F. pulse-rate 124 to the minute. A diagnosis of cerebral compression and meningitis was made, and as a last resort an operation was decided on.

Operation.—The scalp was shaved and cleansed as well as the wound would permit and a tri-radiate incision made. This disclosed a very large and branching fracture of the right side of the skull with slight depression. As there was not apparently sufficient depression to account for the extensive paralysis, the dura mater was incised and the right side of the cerebrum explored with a trocar and then with a finger. Neither blood-clot nor pus was discovered, and the dura was, therefore, stitched together, a tube being left in the cerebrum. The scalp was then brought together and the patient dressed and sent back to the ward.

After-history.—The tube was withdrawn from the brain in a week, and it is worthy of note that no hernia cerebri occurred. The scalp healed slowly, and the paresis soon began to improve. The boy picked up rapidly and regained his normal senses in ten days. He put on flesh and was able to return home on June the 4th, 1908, having still some spasticity and weakness of the left side.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

116. A CASE OF RENAL CALCULUS.

By F. VICTOR MILWARD.

(Introduced.)

The patient, a girl, aged 8½ years, was admitted into the Children's Hospital, Birmingham, on April the 7th, 1908. Her symptoms pointed to trouble in the urinary track, and consisted of periodic discharges of blood with microscopic traces of pus in the urine. These symptoms had been existent for some months, during which time she had never had any local pain or tenderness. While in the medical ward the urine constantly exhibited microscopic traces of pus, together with a few red blood-corpuscles and urate and phosphate crystals. No tubercle bacilli were found, and no stone was detected on sounding the bladder. On April the 30th a skiagram was taken of both kidneys by Dr. Hall Edwards, and the shadow of a calculus was clearly seen in the right kidney and apparently in its upper pole. Nephrolithotomy was performed on May the 8th and the calculus removed. It occupied the pelvis of the kidney, its apparent position in the skiagram being due to a high position of the kidney. Her subsequent progress was uneventful, and she was discharged cured sixteen days after the operation. The cause of the urinary symptoms would have been impossible to verify without the help of the X rays. By them it was shown to be a single calculus and situated in the right kidney.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

117. A CASE OF RUPTURED SPLEEN IN A CHILD AGED EIGHTEEN MONTHS; SPLENECTOMY; RECOVERY.

By F. VICTOR MILWARD.

(Introduced.)

This was a male child who was admitted into the General Hospital, Birmingham, on April the 22nd, 1908, having just been run over in the street by a cart which had passed over his abdomen. The symptoms exhibited were collapse, with great pallor, a very rapid and feeble pulse, a tender and rigid abdomen, constant vomiting and dulness in both flanks. The skin was the subject of severe scabies and impetigo. His general condition on admission was prohibitive of any operation; he was therefore given a rectal injection of saline solution ℥x, brandy ʒj, and carefully watched. Two hours later the pulse had much improved, and with symptoms pointing to intra-abdominal hæmorrhage it was determined to perform an exploratory laparotomy.

Operation.—The abdomen was opened in the mid-line by a four-inch incision extending above and below the umbilicus. The peritoneal cavity was found to be full of blood, and a rupture of the spleen discovered. The tear had extended from the outer border nearly to the hilum. The pedicle was clamped and divided, the lacerated spleen removed and the pedicle then tied. After cleansing the peritoneal cavity the abdomen was closed. The child was much collapsed, and during the operation a venous infusion of normal saline was given by the internal saphenous vein. Shortly after the child rallied well.

After-progress.—But for the fact that the abdominal wound suppurated somewhat seriously the child did well. Between April the 27th and June the 4th five examinations of the blood were made, and it was found to possess a leucocytosis of between 12,000 and 25,000. Much of this was probably due to the presence of pus, but nucleated red cells were also discovered. Fresh spleen pulp or spleen tabloids were given for the first month.

The child had perfectly recovered, and was discharged on June the 12th, 1908.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th*, 1908.)

118. NINE CASES OF MENTALLY DEFECTIVE CHILDREN.

By W. A. POTTS, M.D.

CASE 1.—An extreme example of oxycephaly or *tête à tour*. This case, in addition to great exaggeration of the vertical diameter of the head, shows marked asymmetry of the head and face. Neither of the ears have a scaphoid fossa or a lobule; the right shows a Darwinian tubercle; the left, which is much lower on the head than the right, has a defective helix. There are no eye symptoms. The mental power is not very bad, and improving.

CASE 2.—A typical case of microcephaly associated with complete alopecia in a boy, aged 11 years; the largest circumference of the head is only $16\frac{3}{4}$ inches. Both ears have a large antitragus and very small fissura intertragica. The mental power is very poor.

CASE 3.—A simple congenital case. The boy, though aged 11 years, is only 3 ft. 2 in. high, but shows little other abnormality. The mental power is very poor.

Case 4.—A typical example of macrocephaly in a boy, aged 10 years. The mental power is very poor. The head, which is 22¾ in. in circumference, shows general enlargement, in contra-distinction to—

Case 5, which is typical of hydrocephaly with fair mental capacity.

Cases 6 and 7 are two Mongolians, one or other of which presents all the typical signs. One has a claw nail on the little finger of the right hand.

Cases 8 and 9 are two cretins, one or other of which presents most of the typical signs.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

119. A CASE OF CEREBRAL DIPLEGIA.

By O. J. KAUFFMANN, M.D.

(Introduced.)

A boy, aged 13 years, was exhibited who displayed the following symptoms and history :

Intelligence very good. He has reached the seventh standard at school, and no decline in his mental powers has been observed.

It had become necessary to remove the boy from school, however, owing to an increasing unsteadiness in walking and a growing tendency to fall down, suddenly, and without a stumble, or other apparent reason, for the fall. In one of these tumbles he had broken an arm.

The gait was feeble and halting and limping. The right foot was dragged a little behind the left, all forward progress being made by the latter ; the right foot was slightly of valgus shape, and the sole decidedly flat. In progressing the right foot slightly clung to the ground, but was not extended, nor was there any circumduction. Such falls as had been closely observed were very sudden, and appeared to be due to a yielding of the left knee. After a fall the boy lay still for a few seconds; then rose unaided and without any of the movements which characterise pseudo-hypertrophic paralysis, although the extensors of the hips appeared decidedly a little weak.

On manual testing both the flexors and extensors of the hips and knees were found to be weaker than normal, the feebleness of the right side markedly exceeding that of the left.

There was no wasting of either lower limb; the muscles appeared firm and of normal size in each.

Very little spasticity could be found in either leg, although there was considerable weakness of the foot movements, more pronounced in the right.

Ankle-clonus absent. The right knee-jerk more extensive than normal, but not brisk, and somewhat variable in extent. The left knee-jerk normal. Both plantar reflexes were of flexor type, but the right was duller than the left.

The hands and arms showed no affection, excepting, perhaps, a habit of sitting with the thumb of one hand grasped in the palm of the other. There was no permanent or habitual flexion-attitude of the forearms or hands.

The neck and face appeared perfectly normal.

Respiration was normal, and no difficulty of swallowing had ever been observed.

Lateral nystagmus was present in moderate degree towards each side. Vision was good and the fundi oculorum were normal. No squint.

The whole body was well grown and well nourished.

The personal and family history were as follows: The child was normally born, and began to walk after the age of two years, but had never walked quite normally, the right leg appearing always to be the weaker. The difference in power between the limbs had become slowly and progressively accentuated up to the present time. No mental failure of any kind had been observed.

The patient was an only (and illegitimate) child. Of the paternal history nothing was known; in the mother's family no similar case and no case of paralysis or brain disease in childhood had occurred.

The boy had never suffered from any subacute or acute brain disease, and had enjoyed fair health, being only considered as "rather delicate."

The case was regarded as being, in all probability, one of progressive cerebral diplegia in an early stage, from its early commencement, slow but steady progress, the absence of any wasting, and the peculiar falling. The points against this diagnosis were: (1) The absence of mental change; (2) the very small degree of spasticity; (3) the total escape, after thirteen years, of the arms. But it was considered that, on the whole, the signs and symptoms were in favour of the diagnosis mentioned.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

120. GENERAL HYDROCEPHALUS.

By STANLEY BARNES, M.D.

(Introduced.)

The specimen was taken from a child, aged 3 years, who had been hydrocephalic from birth. All the ventricles are enormously distended, the foramen of Monro being very large.

The condition is apparently due to the presence of adhesions between the medulla and the cerebellum, which have blocked the foramen of Magendie. There was no spina bifida.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

121. A CASE OF PONTINE TUMOUR.

By STANLEY BARNES, M.D.

(Introduced.)

The onset of the illness was with diplopia a few hours after an accident to the head (dropped on to his head by another boy) about nineteen weeks ago. A definite squint was noticed two days later, and this became gradually more marked. Other symptoms have been headaches (moderate), mental deterioration, giddiness, and rolling gait.

He now shows the following signs: Double sixth nuclear paralysis (complete on right, incomplete on left), right facial paralysis and commencing paralysis of the left side of the face, deafness on the right side, but not on the left. No affection of the tongue, but the whole jaw is deflected to the right on opening, owing to paralysis of the right motor fifth nerve. The corneal reflex on the right side is deficient.

There is a considerable degree of left-sided spastic paralysis. The right arm shows intention tremor and is apparently also beginning to become spastic.

The gait is markedly ataxic, and he tends to fall chiefly to the right side.

He had been getting rapidly worse during the last week.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

122. CONGENITAL (? INFANTILE) HEMIPLEGIA.

By STANLEY BARNES, M.D.

(Introduced.)

The brain was taken from a youth, aged 20 years, who died in *status epilepticus*. A few days after birth he was said to have had convulsions, and immediately after this was noticed to have right hemiplegia. In later life the arm and leg recovered some power, but were always smaller than the left, and their movements were clumsy and awkward. He became left-handed, and was sufficiently intelligent to be employed as an errand-boy.

The left half of the brain (shown) is much smaller than the right. The atrophy chiefly affects the parietal convolutions, and the middle of the ascending parietal convolution has disappeared, a cyst surrounded by very small convolutions, and covered at the autopsy by pia-arachnoid, being here present. The ascending frontal convolution is also much atrophied.

The condition was probably due to blocking of the middle cerebral artery, whose remnants have been dissected out.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

123. TUBERCULAR "TUMOUR" OF THE PONS VAROLII.

By STANLEY BARNES, M.D.

(Introduced.)

The specimen shows the brain in sagittal section. A large tubercular mass is seen to be involving the pons and the adjacent part of the mesencephalon.

The patient was a girl, about ten years of age, who in the early stages showed all the signs of disseminated sclerosis: ophthalmoplegia, nystagmus, scanning speech, marked intention tremor of both arms, and later a slowly progressive spastic paralysis of both arms and both legs. Both corneæ became anæsthetic some time before death, but no optic neuritis was present.

A section of the tumour is shown under the microscope.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

124. GLIOMA OF THE CEREBELLUM.

By STANLEY BARNES, M.D.

(Introduced.)

The boy, aged 7 years, came up to hospital with symptoms typical of cerebellar tumour, and an attempt was made to remove the growth. The tumour was not found, and the boy died two months later.

The hernia at the site of the operation is entirely occupied by glioma, which at the time of death had spread upwards so as to block the aqueductus Sylvii and to give rise to a series of metastases in the lateral ventricles by "infecting" the cerebro-spinal fluid.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th*, 1908.)

125. TUBERCULAR "TUMOUR" OF CEREBELLUM, WITH TERMINAL MENINGITIS.

By STANLEY BARNES, M.D.

(Introduced.)

The granuloma was about the size of a hazel-nut, and in the specimen shown half of it has been cut away. The tubercular meningitis is chiefly basal, and is apparently due to direct infection of the meninges from the "tumour."

The specimen was taken from a child, aged 6 years, who was admitted into the hospital with symptoms of meningitis, but who had had headaches and vomiting for some six weeks before. Well-marked optic neuritis was present on admission.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th*, 1908.)

126. OSTEO-SARCOMA OF THE HEART.

By JAMES E. H. SAWYER, M.D.

GIRL, aged 16 years. The patient came under the care of Mr. Gilbert Barling in 1906. She was admitted into the General Hospital, Birmingham, complaining of a swelling of the left knee of four months' duration. The leg was amputated by Mr. Barling on February the 19th, 1906, and the tumour was found to be an osteo-sarcoma.

The patient then went on quite comfortably for about ten days, when she suddenly developed dyspnœa and a cough. The dyspnœa increased, she became very cyanosed, and died from cardiac failure on May the 13th, 1906.

Physical signs.—The heart: The apex beat was in the fifth inter-costal space, just external to the left nipple line. The superficial area of cardiac dulness extended upwards to the third left costal cartilage, and just to the right of the sternum. A rough systolic murmur could be heard all over the præcordia, and was loudest at the apex. The second pulmonary sound was accentuated.

Post mortem.—Secondary deposits were present in the lungs, pleuræ, bronchial glands, and the heart. None in any other organ.

The heart.—There was a small nodule in the right auricle. Large deposits were present in the wall of the right ventricle, some projecting like marbles into the ventricular cavity, so as almost to fill it. No secondary deposit was found in the left side of the heart.

The secondary deposits could not be cut with a knife, but had to be sawn through. They were typical examples of an osteo-sarcoma with small round cells and also spindle cells. The primary and secondary deposits were very similar in character.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

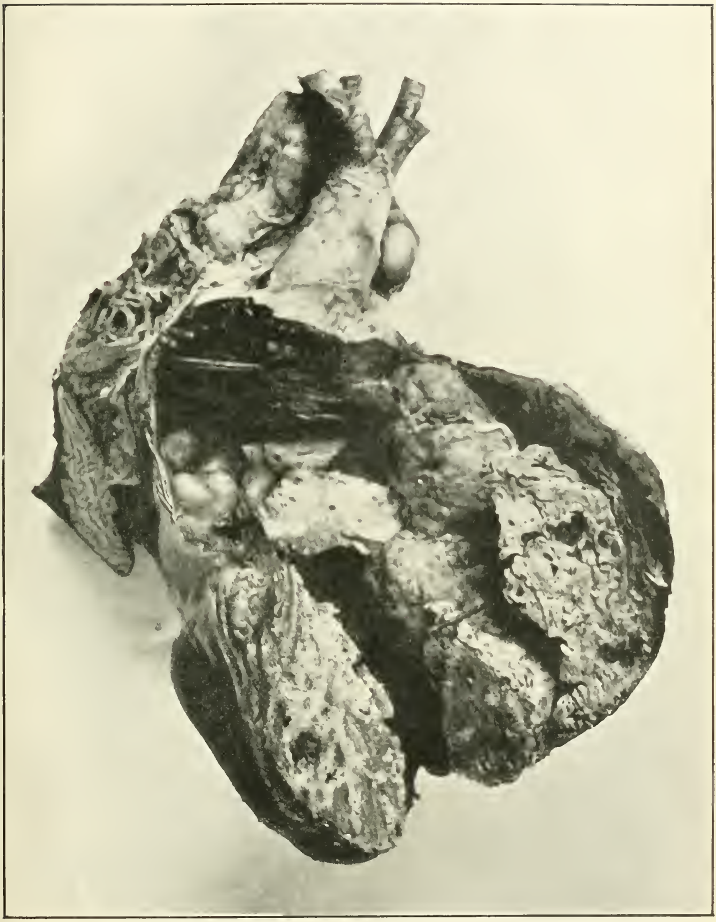


FIG. 63.—Illustrating Dr. James E. H. Sawyer's Case of Osteo-sarcoma of the Heart. Secondary osteo-sarcoma in the walls of the right ventricle and auricle.

127. GLIOMA OF THE RETINA.

By BEATSON HIRD, M.D.

(Introduced.)

These were macroscopic and microscopic specimens of glioma retinæ taken from a child aged 1 year and 9 months. The specimen showed the condition in Stage 2 with secondary glaucoma. There was marked ciliary staphyloma and also staphyloma at the posterior pole. The globe was much distended, the antero-posterior diameter being as much as 33 mm. The growth had extended beneath the choroid posteriorly, but not into the nerve. The retina was detached.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

128. SPECIMENS OF STATUS LYMPHATICUS.

By W. H. WYNN, M.D.

(Introduced.)

Specimens of the organs from several cases of *status lymphaticus* were shown, together with microscopic sections and micro-photographs to illustrate the changes in the thyroid gland which are constantly found in these cases. These changes consist in a marked hyperplasia of the thyroid cells together with a diminution or disappearance of the colloid substance.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

129. SPECIMENS OF TERMINAL RHEUMATIC INFECTION.

By W. H. WYNN, M.D.

(Introduced.)

Organs from several cases of *terminal rheumatic infection in children.* The children had suffered several times from acute rheumatism in its various manifestations and died from a general rheumatic infection involving the endocardium, myocardium, pericardium, pleuræ and lungs. In one case there was a meningitis and in another peritonitis. The so-called "*Diplococcus rheumaticus*" was isolated from the blood in some cases during life and in all from the heart's blood after death, and also from the patches of broncho-pneumonia.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

130. A CASE OF CONGENITAL SYPHILIS.

By DOUGLAS STANLEY, M.D.

(Introduced.)

A child, aged 5 years, the subject of congenital syphilis. The history was typical. The patient showed extensive osteitis of both tibiæ, of the bones of both forearms, and, to a slight extent, of one femur. The teeth were very decayed. There was also choroiditis. The child was admitted into the Children's Hospital, Birmingham, for chronic nephritis which improved under treatment.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

131. A CASE OF SPASMODIC ASTHMA.

By DOUGLAS STANLEY, M.D.

(Introduced.)

A girl, aged 13 years, who had suffered for many years from spasmodic asthma first came under observation when five years old. The attacks were both diurnal and nocturnal. The drug which had the most rapid and satisfactory effect was Grindelia robusta in 15 minim doses of the liquid extract given every fifteen minutes for three or four times during an attack.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

132. EXCAVATION OF LUNG.

By DOUGLAS STANLEY, M.D.

(Introduced.)

A lung from a child who had died of general tuberculosis was exhibited. There were signs during life of excavation, and on post-mortem examination the whole of the right lung was excavated, there being practically no lung tissue left. There were trabeculæ running in all directions and the volume of the organ was much reduced.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

133. CEREBRAL VENOUS THROMBOSIS.

By DOUGLAS STANLEY, M.D.

(Introduced.)

The brain from a child who died of intense anæmia. The red corpuscles fell to below one million; the white cells varied slightly in number. There were retinal hæmorrhages. Previous to death the patient showed irregular paralysis on each side and finally passed into coma. Post mortem, there was marked venous thrombosis over the vertex of the hæmispheres; the brain tissue was soft and red coloured.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

134. CHRONIC ARTHRITIS WITH ENLARGEMENT OF THE LYMPHATIC GLANDS AND SPLEEN.

By N. C. PENROSE, M.B.

(Introduced.)

The patient, a girl, aged 7 years, was admitted into the General Hospital, Birmingham, in May, 1908, and was under the care of Dr. Simon, to whom I am indebted for kind permission to publish the case.

The patient had suffered from swelling and stiffness of the joints for two years. She had never been a strong child, and had always been subject to constipation. She was reported to have had three attacks of pneumonia at the ages of one, two, and three years.

Two years ago her ankles, knees, and wrists began to swell, and at no time since have the swellings diminished in size. There have been attacks of pain in the joints, especially the knees, when it has been most severe.

On admission into the hospital the patient was undersized and very thin. The elbows, wrists, knees, and ankles (Fig. 64) showed marked enlargement. There was no pain and very little tenderness on examination of the joints. There was no bony thickening nor grating. The fingers were becoming affected, especially the thumbs. The cervical spine seemed to have escaped. There was considerable limitation of the movements at the affected

Fig. 64.

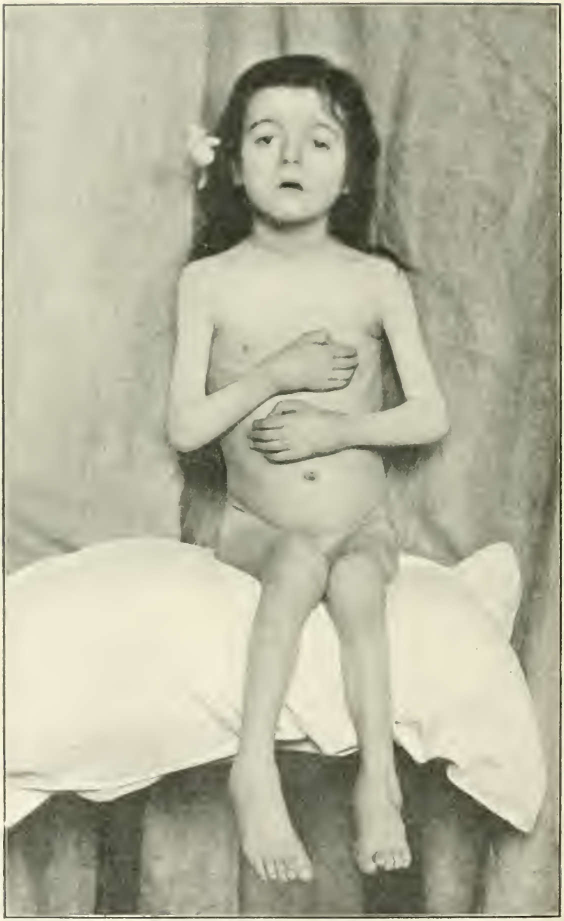
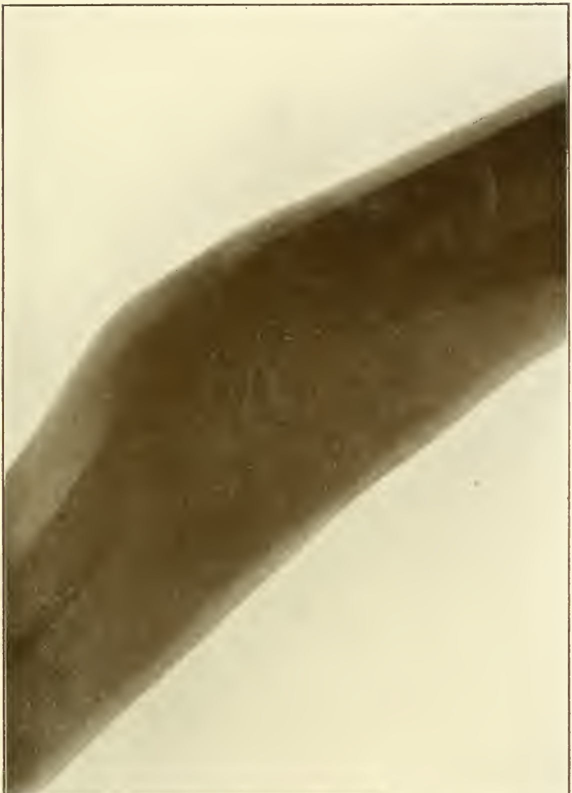


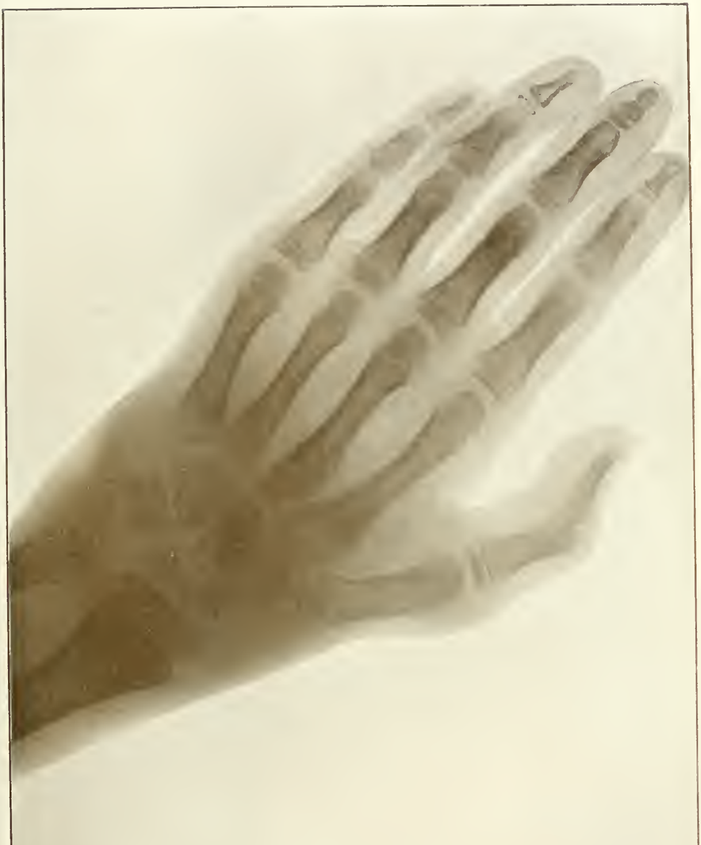
PLATE XII.



Dr. N. C. Penrose's Case of Chronic Arthritis with Enlargement of the Lymphatic Glands and Spleen.

Adlard & Son, Impr.

PLATE XIII.



Dr. N. C. Penrose's Case of Chronic Arthritis with Enlargement of the Lymphatic Glands and Spleen.

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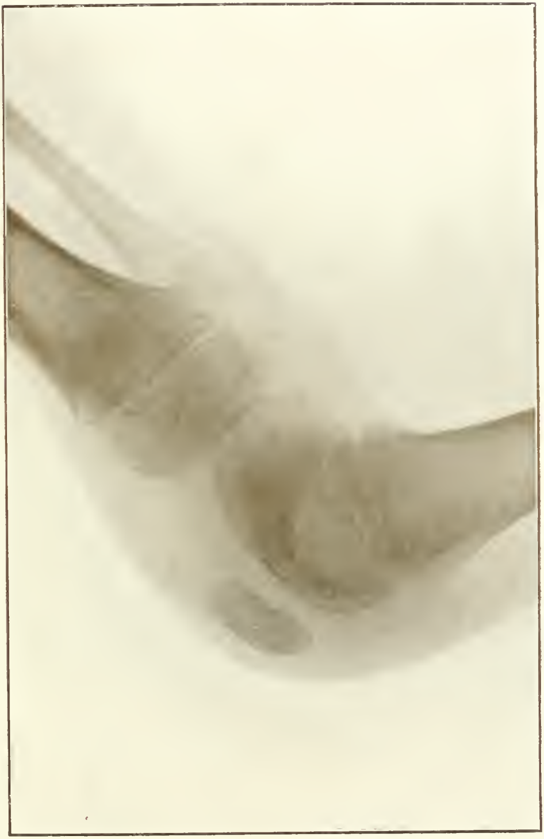
PLATE XIV.



Dr. N. C. Penrose's Case of Chronic Arthritis with Enlargement of the Lymphatic Glands and Spleen.

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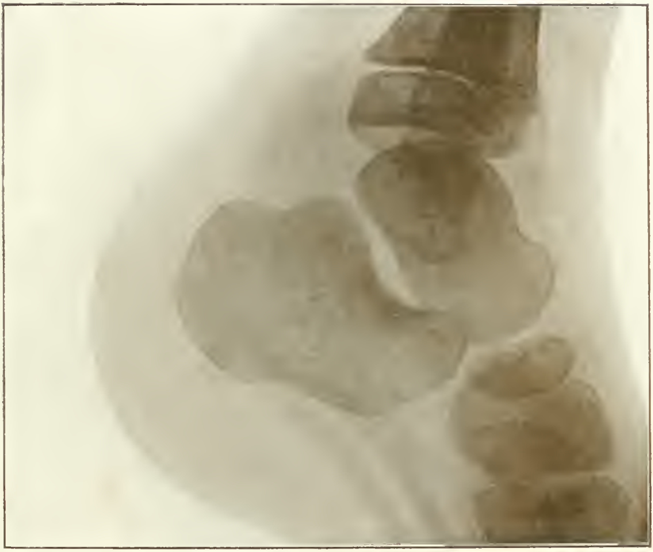
PLATE XV.



Dr. N. C. Penrose's Case of Chronic Arthritis with Enlargement of the Lymphatic Glands and Spleen.

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joints; the muscles of the limbs were wasted, but there were no contractures. The radiographs (Plates XII—XVI), which were taken by Dr. Emrys Jones, showed no sign of bony change, and indicated that the swelling was entirely due to peri-articular thickening. The left wrist was explored with a needle and a very small quantity of fluid withdrawn, which proved to be sterile.

The spleen was moderately enlarged. The lower edge was hard and easily palpable on a level with the costal margin. The lower margin of the liver could not be felt. Nothing else was found abnormal on the examination of the abdomen. The heart appeared normal, except that the apex beat was three quarters of an inch external to the left mammary line. There was enlargement of the sterno-mastoid, supra-clavicular and axillary glands on both sides.

The patient remained in the hospital about six weeks, and when discharged her condition was practically the same as on admission. Her temperature was normal or sub-normal during the whole of that time.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th*, 1908.)

135. HYDROCEPHALUS UNDER TREATMENT BY REPEATED LUMBAR PUNCTURE.

By WALTER JORDAN, M.D.

The head was normal at birth. When four months old the child had a fit, and enlargement of the head was first noticed then. On admission into hospital there was marked hydrocephalus, with gaping fontanelles and sutures and craniotabes. There was marked head-retraction and arching of the spine, with rigidity of the limbs. The child was very fretful; it was unable to swallow and had to be fed with an œsophageal tube. After lumbar puncture the head-retraction and spinal arching disappeared and rigidity diminished. The patient was fed from a bottle, and fretfulness ceased. As the head fills up again the symptoms reappear, and the puncture is repeated before they become really severe. Up to the present eight punctures have been made, the amount of fluid drawn off at a time varying from three to six ounces. After the fifth and succeeding punctures a marked change in the external appearance of the head took place, but the improvement in the symptoms was of shorter duration than after the first four punctures, which had little effect on the external appearance. The circumference of the head has varied from twenty inches before puncture to eighteen and three quarters after.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

136. CONGENITAL PALMAR XERODERMIA.

By WALTER JORDAN, M.D.

Girl, aged 7 years, and her brother, aged 3 years, with marked thickening of the cuticle of the palms. It was first detected by the mother at the age of three months in each case. The father shows the same condition and his father also had it. Two other children have no sign of it.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

137. HYPERTROPHY OF ONE ARM AND THE OPPOSITE LEG.

By WALTER JORDAN, M.D.

Girl, aged 6 years. Very marked hypertrophy of the left upper limb, especially the hand, and of the right lower limb, but scarcely affecting the feet. The hypertrophy is apparently of subcutaneous tissue, and affects the skin of the trunk in the neighbourhood of the affected limbs. The right half of the vulva is distinctly hypertrophied. The condition has been present from birth and shows no alteration.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

138. A CASE OF CONGENITAL OBLITERATION OF THE COMMON BILE-DUCT WITH CIRRHOSIS OF THE LIVER.

By JAMES MILLER, M.D.

(Introduced.)

The child, aged 3 months, was admitted under Dr. Simon at the General Hospital, Birmingham, suffering from jaundice which had been present since birth. The condition was observed immediately after birth. The child never passed any meconium. The bowels had been acting eight or nine times a day, the stools being like curdled milk and very offensive.

The mother stated that she had a shock seven weeks before the child was born. There was nothing to suggest syphilis either in the family history or in the appearance of the child. The liver could be felt one finger's breadth below the costal margin. There was marked emaciation. The child died thirteen days after admission.

At the post mortem the liver was found to be enlarged, of a dark olive-green colour and very firm in consistence. The gall-bladder was firmly contracted and contained no bile. The cystic duct was not patent. The common bile-duct was dilated in its upper part and contained some gelatinous bile-stained material. Four small openings could be seen entering this dilated portion from the liver. The duct was completely obliterated in its lower portion close to the duodenal opening. There were no inflammatory adhesions or thickening round the obliterated portion. The rest of the organs showed bile staining and there was a condition of broncho-pneumonia in both lungs.

Microscopically the liver showed a well-marked monolobular cirrhosis with some invasion of the lobules by fibrous tissue. The liver-cells contained a large amount of yellow and green pigment. In places they were arranged in an acinous manner, the lumen containing homogeneous green material. The fibrous tissue between the lobules was well formed and there were large numbers of small bile-ducts in the cirrhotic tissue. The other organs showed microscopically nothing of note.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

139. A SERIES OF CASES OF BRONCHIOLECTASIS IN CHILDREN.

By JAMES MILLER, M.D.

(Introduced.)

A number of microscopic sections of the lungs were shown from a series of cases of broncho-pneumonia in children, stained by Weigert's elastic tissue method, showing all stages of dilatation of the bronchioles from a condition not visible to the naked eye up to the so-called "honeycomb" lung. The specimens demonstrated that in a large proportion of cases of broncho-pneumonia there occurs a dilatation of the air-passages which are unsupported by cartilage, *i. e.* the bronchioles; that this dilatation is primarily due to a stretching of the elastic and muscular layers of the tubes due to inflammatory infiltration of the walls doubtless assisted by the act of coughing; that this stretching may go on to actual rupture of the laminæ, when large cavities develop filled with purulent exudate giving rise to the condition known as "honeycomb" lung. The reason why such bronchiolectasis is not observed in lobar pneumonia is probably because in the latter condition the smaller air-tubes are supported by the surrounding consolidated lung, whereas

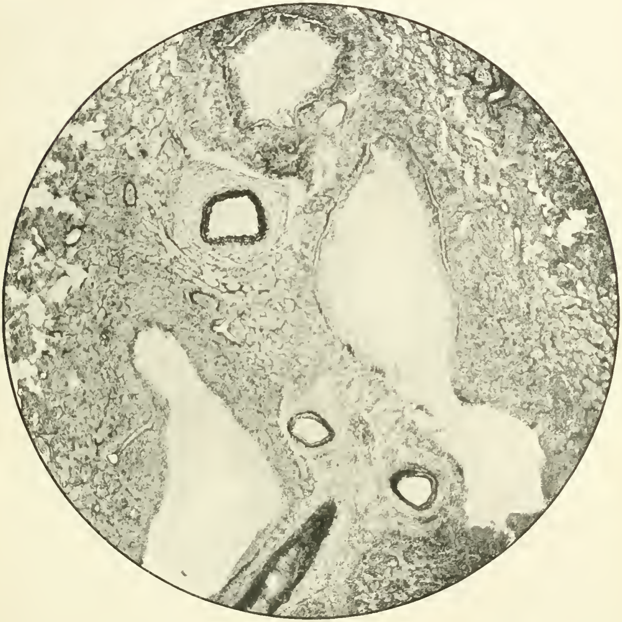

FIG. 65.—Section of lung, broncho-pneumonia, stained by Weigert's elastic tissue method. × 30. Shows above, normal bronchiole with elastic lamina; to right of field another, with the lamina ruptured at one point; below, a third, also dilated, with lamina destroyed.

in lobular pneumonia this is not the case to the same extent.

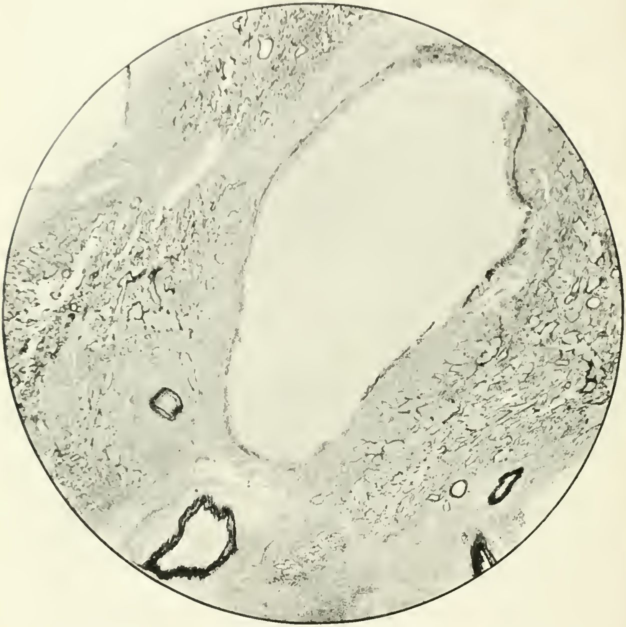

FIG. 66.—Section of honey-comb lung stained by Weigert's elastic tissue method. × 30. Showing enormously dilated bronchioles, with stretched and apparently destroyed elastic laminæ.

(Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th, 1908.)

140. A SERIES OF CASES OF ACUTE TUBERCULOUS CEREBRO-SPINAL MENINGITIS.

By JAMES MILLER, M.D.

(Introduced.)

A number of naked-eye and microscopic preparations were shown illustrating a series of five cases of acute tuberculous cerebro-spinal meningitis in young children with a duration of from two to seven days. In no case were the symptoms typical of tuberculous meningitis, and in more than one a diagnosis was made of acute meningococcal meningitis. Macroscopically there was very little to observe with the exception of an increase of subarachnoid fluid, which was slightly turbid, and some thickening of vessels and nerve roots, but in no case were there any discrete tubercles or obvious caseation. These changes were most marked at the base of the brain and in the upper part of the spinal cord, but spread downwards throughout the whole length of the cord and upwards by way of the Sylvian fissure. In three out of the five cases tubercle bacilli were found microscopically in the exudate; in two of the three they were very numerous. Sections of the brain and cord showed a well-marked lepto-meningitis with fibrinous and cellular exudate, most marked around blood-vessels and nerve roots. The cells were mainly mononuclear in character, although polymorphonuclear cells were fairly numerous. Giant-cell follicles were rarely observed although they did occur. There were no areas of complete caseation, although here and there areas of commencing necrosis could be seen. Tubercle bacilli were found in all five cases, in three of them in large numbers. There was some slight cellular infiltration of the underlying cord, nerve and brain-tissue, and Marchi preparations showed a corresponding amount of degenerative change. In one case no other evidence of tubercle was found although the whole body was examined. In another case there was a tuberculous mass in the right optic thalamus. In a third there was miliary tubercle of all the internal organs without any discoverable point of entrance. In the fourth there were caseous mesenteric glands and in the fifth there was one small caseous bronchial gland.

(*Exhibited at the Provincial Meeting held at the General Hospital, Birmingham, Saturday, June the 20th,* 1908.)

141. ON A LITTLE-KNOWN TYPE OF AMBLYOPIA IN CHILDREN.

By SYDNEY STEPHENSON, C.M.

A CHILD is sometimes brought for advice on account of poor sight, often discovered for the first time during early school life. In the hurry of an out-patient department it may happen that the underlying condition is assumed to be one of ordinary ametropia. The relative visual acuity is probably recorded, and atropine is certainly prescribed for home use. Later, a careful estimation is made of the child's refraction. It is then found that the absolute visual acuity, despite correcting glasses, can perhaps be brought no higher than one third or one half normal, while the optic discs are greyer than they should be. The ophthalmoscopic condition, in short, is one familiar to every physician as "post-papillitic" or "post-neuritic" atrophy. The atrophic changes, nevertheless, are not complete, and may, indeed, be so slight as to escape recognition in the absence of careful ophthalmoscopic examination. In the cases which it is the more particular object of this communication to describe, the appearances are best characterised as those of "incomplete post-papillitic atrophy of the optic disc." They are, so far as I have seen, invariably bilateral, although possibly sometimes more pronounced in one eye than in the other.

Inquiry into the patient's personal history often elicits the fact that when an infant there had been a "cerebral" or "meningitic" illness, marked by such symptoms as fits, convulsions, headaches, vomiting, constipation, retraction of the head, temporary paralysis, squint, unconsciousness, and so forth. On recovery from the attack, sight was found to be partially or totally lost, but has since been more or less regained.

The conclusion is almost inevitable that an infantile meningitis or encephalitis was accompanied by optic papillitis, and that both the general and local condition had to some extent been recovered from. The incomplete optic atrophy, seen later by the ophthalmic surgeon then, is on this view to be regarded as a sequel to the meningitic papillitis.

The type of case indicated rather than described above must be carefully distinguished from two other kinds of temporary blindness, which may occasionally arise under somewhat similar conditions. The first, which I have described in the 'Reports of The Society for the Study of Disease in Children' for 1902, as "Fleeting Amaurosis," and the second "Post-Eclampsic Amaurosis," described by the late Dr. Henry Ashby and myself in the 'Reports' for the following year. The former is met with in infants suffering from basal meningitis, and sight under such circumstances appears to be wholly lost without ophthalmoscopic signs. In the further course of such cases the children often slowly recover both vision and health. It was to this particular class of case that Dr. William Gay proposed to apply the designation "acute cerebral amaurosis of infancy."* The second class presents some points of resemblance to that just described, but the essential cause of the amblyopia appears to lie in convulsions, lasting for some hours, days, or even weeks. When the child regains consciousness he is found to be blind without ophthalmoscopic signs, and as accompaniments may be noted aphasia and hemiplegia of a temporary or of a more lasting type. It will be objected that convulsions are merely accidental complications of many pathological states, and this is doubtless true, but the point upon which Dr. Ashby and I insisted was that the convulsions themselves were the actual cause of the blindness. We suggested in our joint communication that the nerve-storms might involve the visual cortical centres as well as the Rolandic motor centres or the speech centre, and the period of discharge be followed by a period of exhaustion. We published five such cases of what we ventured to call "post-eclampsic amaurosis," in which the patients made a more or less complete recovery.

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* 'Royal London Ophthalmic Hospital Reports,' vol. xiii, 1893, p. 404.

From what has been said it will be obvious that the distinction between the several forms of blindness mentioned in the course of this paper can be established only by ophthalmoscopic examination. In the acute cerebral amaurosis and in the post-eclampsic cases the optic discs show no changes, whereas in the other class they exhibit incomplete atrophy. The distinction, then, can be difficult only when a child is so young as to resist anything like a full ophthalmoscopic examination of the eyes.

CASE No. 1.—*Imperfect sight in a girl, aged* 11 *years, due to an attack of meningitis, or encephalitis, entailing optic atrophy* (*incomplete*) *four years before she fell under notice.*

Helena C—, aged 11 years, brought to the Ophthalmic Department of the Evelina Hospital on November the 26th, 1907, on account of poor sight. No personal or family history of syphilis. No family history of defective sight. R.V. = $\frac{6}{12}$ and No. 1 Jaeger. L.V. = $\frac{6}{12}$ and No. 1 Jaeger. Pupils 4·5 mm., responsive to light (direct and indirect action) and to convergence. Fields for white showed insignificant concentric contraction. A week later, the eyes then being under the influence of atropine, with the total hypermetropia corrected (0·5 D.), the vision of each eye could be brought to $\frac{6}{9}$ letters. There was post-papillitic atrophy of each optic disc, somewhat more marked on the right than the left side.

A history was obtained to the effect that at seven years of age the child suffered for some seven or eight weeks from very severe headaches, as well as from bouts of vomiting, and that she was ill in herself.

CASE NO. 2.—*Amblyopia, nystagmus, and feeble-mindedness in a lad, aged 10 years, in whom there was a history of meningitis at two years of age.*

George B—, aged 10 years, admitted into the Queen's Hospital for Children on April the 9th, 1908. Although he might be described as "feeble-minded," yet he was clean in his personal habits and not viciously inclined towards other children. R. V. = $\frac{5}{60}$ and No. 12 Jaeger. L. V. = less than $\frac{5}{60}$ and No. 12 Jaeger. Pupils equal and active to light. Slight lateral and rotatory nystagmus. Refraction (estimated under atropine): R. V. = $\frac{+\ 0{\cdot}5}{+\ 1{\cdot}5\,90^\circ}$. L. V. = $\frac{+\ 1{\cdot}0}{+\ 1{\cdot}0\,1}$. Optic discs in a state of incomplete post-neuritic atrophy. Their colour was dirty greyish-white, their edges a little ragged, and the lamina cribrosa was not visible. Knee-jerks plus. No ankle clonus. ? Babinsky. No ataxy. No stigmata of hereditary syphilis. The history was as follows: At two years of age, one night the child developed what was described as a "screaming fit," and on the next day there was a definite "fit," which lasted twenty minutes. Since that time the lad has been subject to "shivering fits" and to "facial distortions" every few months.

CASE NO. 3.—*Amblyopia, nystagmus, squint, and feeble-mindedness in a child aged 7 years, who three years previously had suffered from meningitis.*

Albert H—, aged 7 years, was first seen at the Ophthalmic School, at Hanwell, on February the 21st, 1893. He was bad-tempered and feeble-minded, and his speech was difficult to understand. Nystagmus was present. The sight was defective, but the lad was certainly able to count fingers at a distance of twenty feet. Both optic discs were incompletely atrophic. Upon inquiry it was ascertained that about three years before the lad came under my notice, at a time when he was about four years of age, he complained of headache, lost the power of walking, and developed a marked tendency to vomit. His head was held stiff, his left eye squinted inwards, and there was a discharge from his right ear.

The lad remained under my observation until he was twelve years of age. On July the 18th, 1898, the sight in his right eye was equal to about one half normal and that in his left eye to about one fifth of the normal. The left eye tended to diverge. Fixation was imperfect. Colour-blindness was present. The state of the optic discs was as noted previously. What I took to be a large haemorrhage undergoing metamorphosis was present in the central region of the left fundus oculi.

CASE No. 4.—*Incomplete optic atrophy and convergent squint in a young child who three months previously had been affected with an illness diagnosed as meningitis.*

Arthur J. M—, aged 18 months, was sent to me on January the 18th, 1894, by Dr. C. E. Adams, of West Norwood. The history was that three months before coming under my notice the baby became ill, was drowsy, and did not care for his food. The condition gradually became worse. The child suffered from pain in his head, was convulsed five or six times a day, his neck was stiff and his head retracted. There was, however, no vomiting. The right eye squinted inwards. The sight, which was good before the attack, seemed to fail in the third week of the illness. The latter, diagnosed by Dr. Adams as meningitis, lasted about two months and a half. The patient was the sixth child, and was born at term after a natural labour lasting twelve hours. When I examined Arthur J. M— he was found to be fairly well-nourished, with all his teeth except two. The anterior fontanelle was not widely open. The right eye had a large convergent squint, seemingly due to paresis of the external rectus muscle. The pupils dilated readily to homatropin. The optic discs were sharply defined and somewhat white. The child retained just sight enough to move about without running into the furniture.

CASE NO. 5.—*Nystagmus, squint, and incomplete post-papillitic atrophy of the optic discs in a child, aged 21 months, seen five months after an attack believed to be one of meningitis.*

Marjory B—, aged 21 months, first seen on May the 6th, 1908. She had been healthy until towards the end of the first week in December, 1907, when she was convulsed for twenty-four hours and became "stiff." Power was then lost—first on the right and then on the left side of her body. No vomiting. The head was markedly retracted. The baby was "quite paralysed" for two weeks. Her temperature was 105° F. on end for several days. Enemata were necessary on account of obstinate constipation. Inflammation of both lungs and bronchitis then supervened and lasted for seven days. During recovery from pneumonia the child is stated to have gone completely blind, but this symptom probably preceded the onset of the lung mischief. After the illness had lasted for some weeks the baby then began to recover her sight, the first object she saw being a tan shoe lying at the foot of her bed. Further improvement took place during the voyage from the Cape to England, which began on April the 6th, and has continued since arrival in this country towards the end of April. The baby's eyes were first noticed to squint and to roll about when sight began to be regained. On examination, pupils 3 mm., with but slight response to light. Slight rotatory nystagmus. Optic discs pallid. Notices objects fairly well. Child walks fairly and talks well. Power over legs (lost during illness) almost regained.

Remarks.—The cases show that an attack of meningitis, or encephalitis, may entail as permanent evidence of its former existence: (1) Incomplete optic atrophy; (2) nystagmus; and (3) paresis of one or more of the extrinsic muscles of the eyeball. The symptoms, it will also be seen, may occur as isolated or grouped phenomena. In the last case to be quoted, paresis of extrinsic muscles was the only evidence of a former meningitis.

CASE NO. 6.—*History of meningitis at six months, followed by paresis of abduction in both eyes.*

Donald W—, aged 6 years, brought on June the 2nd, 1906, on account of defective sight. Vision was almost normal on correction by suitable glasses of the compound hypermetropic astigmatism that was present. The fundi showed no departures from the normal. The movements of the external recti muscles were obviously defective, so that the outward excursions of the eye were neither full nor well maintained. On seeking for a cause for this anomaly, which clearly might represent a congenital defect, it was ascertained that when about six months old the child had been ill for three weeks, suffering from vomiting, incessant moaning, rocking movements of the head, and a high temperature (over 105° F. on several occasions). His father, a medical man, thought the attack was one of meningitis, a view concurred in by another experienced practitioner who was called in. But when recovery took place the symptoms were put down to influenza.

In conclusion, I desire to state plainly that the present communication lays no claim whatever to originality. The existence of cases such as those I have described has been known for many years to some observers, although, so far as I am aware, they have not yet found their way into text-books, whether of general medicine or of ophthalmology. For example, Mr. Edward Nettleship published in 1883 a communication "On Cases of Recovery from Amaurosis in Young Children."* Of the nineteen cases of which notes were given in that important communication, Nos. 6 and 9 appear to belong to the group I have ventured to describe on this occasion. In a later contribution* the same distinguished observer re-quoted two of the cases alluded to in his former paper.

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* 'Ophthal. Soc. Trans. of the United Kingdom,' vol. iv, 1884, p. 243.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th,* 1908.)

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142. ON TWO CASES SUGGESTING RELATIONSHIP TO STATUS LYMPHATICUS.

BY OTTO KAUFFMANN, M.D.

(INTRODUCED.)

THE cases which I am bringing to your notice are examples of Hodgkin's disease and of splenic anæmia, and I wish to elicit a discussion on the relationship between status lymphaticus and those diseases. The first case is that of a school-girl, aged 12 years, tall, slim, pale, but well grown, very intelligent and with fairly good muscles. She entered the hospital in the early part of this year. There were enlarged lymph-glands on both sides of the neck, both supra-clavicular regions and both axillæ, but none in the groins. The glands were of the size of hazelnuts and discrete, except in the right axilla, where a large soft swelling underlay the bulging pectorals, and obscured the individual lymph-glands. There was enlargement of the area of splenic dulness, and when she first came into hospital the spleen was doubtfully palpable, but it soon became obviously so. It, however, never exceeded two inches below the left costal arch. She had no internal pressure signs; the blood looked pale, but showed no marked departure from the normal on microscopic examination.

I think the case may fairly be called one of Hodgkin's disease. The teeth in both jaws were very carious, and the breath smelt foul. Her temperature was not steady; it rose to 101° F. and sometimes even to 102° F., but for a week before her discharge in April the temperature was again normal. Except for the improvement to her general health from rest and good food I did not trace any benefit to treatment. The glands did not vary much in size. She did not react to the Calmette test, and there were no signs suspicious of tuberculosis in any part of the body. From February the 27th to March the 11th she was taking iron continuously, but neither the hæmoglobin nor the red cells improved. The counts* were all made at the same hour of the day. From March the 11th to the 31st she was on liquor arsenicalis 5 ♏ three times a day. Her red corpuscles then rose a little, the white ones fell a little, and the hæmoglobin improved. From March the 31st to April the 22nd she had mercurial inunctions, and the white corpuscles fell a little as also did the red ones. The treatment was changed from arsenic because her general health did not seem so good as I hoped would be the case under arsenical medication.

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* 'Royal London Ophthalmic Hospital Reports,' vol. xi, Part IV, July, 1887.

Blood Counts of the First Case.

	Red blood-corpuscles. Per c.mm.	White blood-corpuscles. Per c.mm.	Hæmoglobin. Per cent.	
Feb. 29	3,600,000	9,000	65	Feb. 27—March 11, iron.
March 11	3,200,000	14,000	65	March 11—31, liquor arsenicalis ♏v.
April 7	4,370,000	8,000	70—80	March 31—April 22. unguentum hydrargyri.
,, 11	3,500,000	6,000	70	
June 16	3,760,000	13,408	75	

The other case was that of a girl, aged 21 years, who died. Her case might be regarded either as one occupying an intermediate position between splenic anæmia and Hodgkin's disease, of the type associated with enlargement of the spleen, or as one of Hodgkin's disease proceeding to severe anæmia. Multiple hæmorrhages and an acute inflammation of the larynx with œdema, necessitating intubation, set in, finally killing the patient by exhaustion. The first thing noticed was enlargement of the spleen, which, even on admission, was very big, and which, post mortem, reached across the mid-line and down into the false pelvis. After death many petechiæ were found on the heart and pericardium. The thymus was normal, but the lymph-glands all over the body were greatly enlarged, without caseation, although in the lungs there were old tubercular foci. The liver was congested, with round-celled infiltration of the interlobar spaces. The spleen was enormous, and there was much perisplenitis. The kidneys showed round-celled infiltration, particularly of the cortices. There was poikilocytosis, but there were no macrocytes. The blood count on admission was $2\frac{1}{2}$ millions red corpuscles and 7000 white corpuscles per c.mm. The hæmoglobin index was slightly over 1. The treatment by arsenic showed only an evanescent improvement. None of the drugs tried materially altered the condition of the blood.

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* See table.

Blood Counts of the Second Case.

		Red blood-corpuscles. Per c.mm.	White blood corpuscles. Per c.mm.	Hæmoglobin. Per cent.	
Feb.	4	2,560,000	7,190	80	Feb. 3—9, Liq. Fowleri ♏v.
,,	17	2,800,000	8,100	—	Feb. 9—March 1, Liq. Fowleri ♏x.
,,	26	3,300,000	6,000	—	—
March	1	3,100,000	6,600	85	March 1—27, iron and arsenic.
April	1	—	2,600	—	March 27, nuclein.
,,	4	—	3,200	—	—
,,	5		4,200	—	—
,,	7	—	4,800	—	—
,,	8	2,568,000	3,200	60	—
,,	13	—	4,800	—	—
,,	17	—	5,400	—	—
,,	22	2,552,000	7,200	60	April 22, nuclein stopped.
May	4	1,860,000	7,400	50	—
,	8	—	9,600	—	—
,,	19	902,000	14,400	—	—

My object is to draw attention to the very probable connection between these cases and the condition which has been called status lymphaticus, or status thymicus. Probably members have seen at the General Hospital the beautiful specimens which Dr. Wynn had placed there, showing the changes met with in that disease. I should like to discover whether it is the general experience that the status lymphaticus is apt to develop into Hodgkin's disease or some other form of pseudo-leukæmia. I suppose that in all cases the definition of status lymphaticus involves necessarily a hypertrophied thymus. There was no such hypertrophy in the fatal case. Still, it presented the general picture of that disease, and I cannot help thinking that there is so intimate a relationship that a case of status lymphaticus might pass into one of Hodgkin's disease, and that, as the second case shows, that condition may proceed to intractable anæmia.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th*, 1908.)

Discussion.

Dr. F. H. Haynes (Leamington) (introduced) wished to call attention to the value of mercury in some of these cases of lymphadenoma. Long before there was any discussion on blood counts, a girl, aged 15 years, came to him with a large mass of glands on the left side of the neck, so large that the surgeon declined to operate on them. The enlargement went on until the tumour was of the size of a cocoa-nut, and the other side under treatment was also very big. He put her on perchloride of mercury, which she continued taking for three years, beginning with one sixteenth of a grain and going up to one fifth of a grain three times a day. Both glandular masses entirely disappeared, and she was now alive. It showed in a remarkable manner the value of mercury, and that it might be continued without producing symptoms of ptyalism. He believed the perchloride seldom salivated. He could trace no history of syphilis in either parents. Her mother died from hemiplegia at the age of 59 years, and her father was now in the lunatic asylum, aged 75 years: the latter had Argyll-Robertson pupils.

143. SOME FACTORS IN THE CAUSATION OF THE NEUROSES.

By WALTER JORDAN, M.D.

In making these brief remarks on the causation of the neuroses I have in mind the general neurotic condition to be observed in so many children, characterised by restlessness, timidity, irritability, tearfulness, moral delinquencies and such rather than the more definite neuroses such as enuresis, asthma or tetany. Whatever circumstances lead to the development of these states must obviously be either pre-natal, natal, or post-natal. Accidents of birth cause gross lesions rather than neuroses, while of pre-natal influences all but a very few are hereditary. I wish to speak first of the hereditary factor in the causation of the neuroses, and, to come to the point at once, to ask whether we are not inclined to attribute too much importance to it, to regard its presence as proved often on very slender evidence, and even to assume it where there is no direct evidence whatever of its existence. The better educated of the laity are absolutely obsessed by the hereditary explanation of nervous and mental affections, and a considerable section of the medical profession is at one with them on the point. I do not want to be misunderstood; I admit the supreme importance of hereditary influences in many cases of neurotic children. It were foolish to go to an opposite extreme and refuse to credit the theory of ancestral failings at any time, but one may ask, is it not overdone, and—for here is the practical point—has it not sometimes so engrossed attention that post-natal circumstances, still active, and possibly capable of modification, have been overlooked, with the result that treatment has had to remain vague and general, when it might have been direct? For the essential drawback of the hereditary theory of causation is that while it emphasises the necessity for careful treatment it supplies no clear indication how to treat. When we are face to face with a neurotic child, the statement that treatment should have begun two generations back, however picturesque as phrase making, is discouraging as doctrine. In support of my case I would point out two things; first, that it is not always easy to say whether certain parental attributes are to be regarded as factors of heredity or of environment. A neurotic child has a drunken father. Would it have been neurotic had it been taken from him when a week old? What of the anxiety, the fear, the broken sleep, possibly the physical injury? Or a neurotic mother has a neurotic daughter; again, what might have been the effects of another than the maternal upbringing? The second point is that, even with fairly intelligent parents, it is by no means easy to get a trustworthy history of the infancy of the children of 5 or 6 years of age whom they bring to you. Even if they observe them they do not know the significance of the ailments of babyhood. So much of what happened in the first eighteen months of life is lost to mind, and the origin of many a neurosis is "wropt in mist'ry." Two questions may be put: the one, Is the nervous system of any child so congenitally defective that, in spite of proper treatment and education from the first, the child must grow up neurotic? The answer to which I believe to be, Yes, but apart from actual imbeciles, of very few. Next, Is the nervous system of any child so congenitally perfect that no illness or error of training can render it neurotic? The answer to which I believe to be, No. Both these answers, if correct, emphasise the importance of the post-natal causes of neurosis, turning to which I select two as being extremely common, and often overlooked or under-rated. The first is habitual constipation in infancy or childhood. We ourselves are well aware of the evil effects of this condition on the nervous system amongst others; but the difficulty is to get parents to recognise that it is a condition which calls for careful continuous treatment at skilled hands, instead of the spasmodic domestic administration of drastic purgatives or inefficient laxatives. I fear that occasionally even members of the profession, when the point of a youngster's habitual constipation is referred to them, instead of insisting on the necessity of a deliberate and ordered campaign of treatment have been content with a hasty recommendation to try something or other. The condition favours the development of neuroses, not only directly by the action of toxins absorbed from the alimentary canal on the neurons, but in many indirect ways. Thus the constipation of breast-fed babies too often leads to their being taken from the breast under the misapprehension that their discomforts are due to the quality of the breast-milk. Launched, then, on the perilous sea of artificial feeding, they are exposed to the further risk of starvation of the neurons from an insufficient supply of building material. Or again, pain may prevent sleep, and continued loss of sleep is in itself enough to account for much neurosis.

Habitual constipation is generally known to be present, even when it does not receive the attention and treatment it deserves, but there is a causal condition of neuroses which is quite frequently entirely overlooked. I refer to septic tonsillitis of the follicular or lacunar type, which for some reason or other, even when it causes marked enlargement of the tonsils, frequently gives rise to no pain or difficulty in swallowing, with the result that no complaint of sore throat is made by the child and the condition goes untreated. It is quite common in infants, who are, of course, unable to point out the seat of mischief. What does arouse attention is the accompanying adenitis, affecting glands about the angle of the jaw. When this glandular enlargement is great it is not uncommonly mistakenly diagnosed by parents as mumps, and an expectant attitude adopted. If the adenitis is slight or escapes notice, the only thing that will lead to the child's being brought for treatment is severity of the ensuing general debility or the appearance of some more definite sequela, which may be a functional nervous disorder such as enuresis. No condition is more liable to become chronic or more prone to recur than this variety of tonsillitis, and the injury done to the general health of children by it is incalculable. It is at the back of a great deal of the nervousness, naughtiness, and irritability for which so many children are brought to us that we may say whether "they can help it," or whether there is something really wrong with them.

I do not propose to deal with any other of the post-natal causes of neuroses. Rickets and rheumatism as causes of neuroses in childhood are well known to every one of us through literature and from personal experience. They suffer from no disregard. Over-pressure in education is also the subject of vigilant inquiry; even in infancy over-pressure has been reported as a cause of a neurotic condition where parents, over-proud of the way in which their babe takes notice, have absolutely exhausted it with an incessant stream of more or less dazzling stimuli. And there are yet other causes. But I have selected these two, because I believe that in the one case the invasion of toxins from the throat is quite frequently overlooked, and that in the other the toxic and other dangers of habitual constipation are too often left unguarded, and that never to omit investigation of the condition of the throat, and never to scamp the treatment of constipation in neurotic children, would lead to more practical benefit than time expended in minute inquiries as to whether a great aunt of the patient did not suffer from migraine or a mother's cousin from religious mania.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th,* 1908.)

Discussion.

Dr. Charles W. Chapman said he was sure that constipation and tonsillitis often went together, and if the patient were promptly given a purge, frequently the throat would get better. In children constipation many times had an effect on the nerves; they often had what was known as diarrhœa, which he attributed to lumps in the colon, which acted as irritants in the same way as did a grit in the eye. It was not necessary to go far for evidence as to the effect of constipation in producing low spirits and a gloomy outlook; the pessimistic psalms would show that. If the writer of these psalms had had a blue pill and a black draught twice a week these psalms would probably not have been produced. He had now under care a child which had been the victim of over-pressure in school matters and improper feeding. Although living at the seaside she was kept indoors nearly the whole day, and had special lessons, succeeding each other practically without a break. The child had a white face and a dilated stomach, and was as jumpy and nervous as possible. He regarded the paper as a very valuable one.

Dr. George Pernet said he agreed with the author that heredity was overdone. Heredity had been given up as regards tuberculosis and leprosy. He was satisfied that syphilis was not strictly an inherited disease; in his opinion congenital syphilis was of maternal origin.* With regard to neuroses, was it any wonder that children were neurotic when the way in which they were brought up was considered? Some years ago a little girl, aged 11 years, was brought to him as he happened to be a friend of the father. This particular child was trying to keep up with the other children of the class, on the average two years older than herself, and was being taught a number of "ologies," etc., including, strange to say, physiology! The great part played by environment in the production of neurosis was apparently overlooked, especially by amateur teachers of physiology.

Dr. Edmund Cautley said no doubt most of those present would agree with the views put forth by Dr. Pernet if skin diseases were meant, but he doubted whether the term was as much abused as Dr. Pernet seemed to think. He, Dr. Cautley, did not doubt that heredity played a very important part in the production of a large number of diseases, more especially so-called neurotic affections. There was an old saying that one could not gather grapes from thorns nor figs from thistles. If there was a strong neurotic heredity there was likely to be a neurotic child, even apart from such complications as constipation or lack of fresh air and sunlight. It would be dangerous to attach too much importance to any one factor in the production of neurosis, because neurotic children were met with under the most perfect external conditions and with a good inheritance. The most robust country children, apparently, would be found to develop habit-spasm, and that could not be attributed entirely to local surroundings. On the other hand, children living under the most insanitary conditions, who had alcoholic parents, or even epileptic or insane parents, and who lived in a confined atmosphere, would develop into normal healthy adults. In fact he was inclined to regard constipation as a sign of the torpid rather than the neurotic child. He did not attach much importance to constipation in those cases, but otherwise he agreed with the views the author had advanced.

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* Pernet, on "Congenital Syphilis," 'British Journal of Children's Diseases,' 1908.

Dr. Walter Jordan (Birmingham), in reply, said he agreed with Dr. Cautley that too much importance must not be attached to any one factor in the causation of neuroses; it was only because he thought too much stress was laid on heredity as a cause that he had contributed the paper.

144. THE PROGNOSIS OF HEART DISEASE IN CHILDREN.

By DOUGLAS STANLEY, M.D.

(Introduced.)

One of the most important problems that confront us in practice is the future of a child who has a mitral valvular lesion.

There are many factors on which this future depends, as, for example, the social position and the intelligence of the parents or guardians—in a word, the whole of the circumstances under which that child has to live and grow. But there are others which are less tangible, and which we cannot always estimate at the time—factors whose value can only be gauged in a number of years.

I have watched the course and progress of many cases of heart lesion, especially mitral, in children for several years. The results have been for me very important and instructive. In some of these cases I was able to see the beginning of the process, that is, to have the patient under observation at the onset of the heart lesion—a point of the utmost importance obviously. In others it was the common story of finding a valve defect, already established in a child brought on account of debility or any of the various symptoms that indicate heart lesion in children, and where we have less chance of doing good than in the former group of cases.

The following case, the boy Martin, aged 11 years, whom you have seen, is an interesting example. Nearly two years ago he was brought to me at hospital on account of heart pain; he appeared very ill. I found loud pericardial friction, and at once took him in. A large effusion rapidly appeared—so large that I had to tap, as the boy showed all the worst indications of cardiac compression. He made an uninterrupted recovery. No lesion could be detected afterwards. He came in about a year later with acute rheumatic symptoms. This time a soft systolic murmur could be heard, and the general cardiac signs made me fear that considerable damage might result. The boy was kept absolutely at rest for two months. Since then he has been very carefully looked after. He has been very little to school and all violent exercise has been forbidden. As you saw him to-day, he looks a bright and active boy. The heart condition is very satisfactory. The impulse is in the fifth space, half an inch inside the mid-clavicular line. The mitral first sound is a little fluffy, there being what can hardly be called a murmur, and what there is, is soon soon lost outside the mitral area. The second is accentuated and reduplicated. The tricuspid and aortic sounds are good average quality. The pulse is regular and of good size. No veins pulsate in the neck.

This boy is, as I said, very carefully looked after, and for some years to come this supervision will be continued, for fortunately his mother is an intelligent woman, who is willing to do as advised. Given freedom from further rheumatic infection and any other serious illness this boy should, and I believe will, so considerably improve as he grows that possibly his heart may show little, if anything, of past lesions.

A boy, the son of a doctor, was brought to me about six years ago because his father discovered a mitral murmur. The patient was not a full term child, and had a delicate babyhood. When about six years old he ailed for a time, though there was nothing very definite or suggestive of acute rheumatism. I found he had mitral incompetence, there being a well-marked musical systolic murmur with accentuation and reduplication of the pulmonary second.

There was in addition to the drawback of a not robust constitution the question of school in the immediate future. My advice was to get the child to grow and strengthen as much as possible; when sent to school, it should be to some place where he would be looked after and not allowed to overtax his strength. As far as possible there was to be a period of daily rest on his back. This plan was followed for several years. He grew and strengthened, and though now not a strong-looking boy, he is not in appearance much below the average. Quite recently—that is, nearly seven years after my first examination—I was asked to see him again to decide the question of greater latitude in school games. The cardiac condition had entirely altered. The heart's action was regular, the mitral first sound was well marked, and I was very doubtful if anything suggesting a murmur could be heard. Yet in this case a well-marked musical systolic was present for a considerable period—over two years.

The child Osborne, aged 11 years, whom you saw at the Birmingham General Hospital this afternoon, was under my care in hospital on account of well-marked mitral regurgitation, the result of acute rheumatism. I am told that a doctor who treated her at home said she had pericardial effusion. I first saw her about three years ago. She has grown since then. To-day her cardiac impulse is practically in the mid-clavicular line. The mitral first sound is loud and vibrating, accompanied by a slight systolic murmur, which is not heard outside the mitral area; it is somewhat increased by quick movements. The pulmonary second sound is reduplicated and accentuated. The pulse, though of fair volume, is not always regular. This patient has shown so much improvement that I hope there may be still more, and that as she grows there may be corresponding change in the condition of the heart.

I think we are inclined to make up our minds concerning the future at too early a period in the history of heart lesions in children. Certain I am, from watching some of these cases for a number of years, that great improvement may, and does, take place. I believe that, when the lesion is mitral, that if circumstances are favourable—that is, social surroundings and the parents able and willing to follow directions for a long period, without forgetting the past—the heart participates in the general growth of the child and leaves much of its past behind it. If, however, the lesion be a severe one with much valvular deformity, if there be excessive hypertrophy, especially if there be any pericardial adhesion, or, I may say, if there be reason to suspect pericardial adhesion, the outlook is bad. For instance, I had a little girl in Queen's Hospital some months ago for well-marked mitral disease with double murmur and considerable dilatation. In a sense she did very well, and left the hospital comparatively comfortable, there being fair compensation. But here I hope for little. The child looks badly; from her slightly "mitral" appearance—I can hardly call it cyanotic—it is evident that her vascular system is embarrassed. She is not, I think, growing, although she has put on some weight. Physical examination shows pronounced hypertrophy of the heart, and a marked systolic mitral murmur. It is difficult to say exactly what we mean by a severe mitral lesion, but when I find that the murmur varies under observation, especially if a shifting diastolic be present as well as a systolic, I regard the lesion as severe. In the case just quoted these points are noticed.

I have referred to the question of growth. If a child with cardiac valvular disease grows, we may say there is a better chance for the heart than if he remains undersized and puny. I do not think this has been sufficiently emphasised. We are often asked the question by parents:

"Will he grow out of it?" I think we may in some cases of valvular disease say it is possible. It comes to this, and here is my point, that if we can so order a child's life for a period of years, which must extend past puberty into adolescence, if not manhood and womanhood, that everything will conduce to steady growth without strain on the cardio-vascular system; if, with luck, there be no further damage from infections, we may hope for disappearance of a valvular lesion. The period of puberty requires particular attention, and it is just at this time, in deference to what we call civilisation, that the strain of education is greatest. It is then that there should be the greatest care, and a period of daily rest on the back to compensate for rapid growth and the great cardio-vascular changes.

It is most interesting to watch these cases during such a crisis. About ten years ago I saw a child with undoubted mitral disease. Though the history of actual acute rheumatism was doubtful, there had been bad attacks of tonsillitis. I gave a bad prognosis. I saw her at intervals for some years. When about sixteen she was a fairly well-grown girl, and no lesion could be found in the heart. It was suggested when I showed her at one of the Medical Societies that the lesion was what is called functional, because it had disappeared. All I can say is that if it were, then many cases of valvular lesion with marked confirmatory signs are functional, a position I am now less inclined to accept, in view of many other cases I have watched.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th,* 1908.)

Discussion.

Dr. Charles W. Chapman said it was very cheering to hear so hopeful a view taken of these cases. He had felt very strongly that to say the prognosis in a child with valvular lesion was necessarily bad was wicked, as many of such cases did wonderfully well. He had a patient, an old lady, who had a presystolic murmur dating from rheumatic fever at fifteen years of age, who had enjoyed quite average health. He had seen congenital cases where the physical signs disappeared after a few years. What the exact condition of the heart was in these recovered cases could only be conjectured, as they did not come to the post-mortem table. Moreover, congenital cases frequently improved under treatment, showing there was no justification for an universally gloomy prospect regarding them. A child with aortic regurgitation, if it grew fast, would have faintness due to cerebral anæmia, which rest and subsequent hypertrophy would compensate for. Also in the case of a child, the parents as well as the patient had to be taken into consideration in giving a prognosis. A bad prognosis deprived the parents of hope, and gave them the idea that all treatment would be unavailing. Even if treatment would only prolong the life for three or four years, it was an important matter for the parents.

Dr. Porter Parkinson said he thought Dr. Stanley had brought forward a very important point in emphasising the growth of the child. There was no question in the minds of those who followed up their cases that, in severe instances, especially of mitral stenosis, stunting of the growth was very common, and it would be useful to make a record of the child's weight and height every three months to ascertain the extent of its progress. As to the examination of the heart itself, he always thought that inspection and palpation were most valuable methods for arriving at a prognosis. The mere fact of there being a murmur in one or other situation seemed to be of comparatively little importance compared to the condition of the heart muscle. It was probably owing to the malnutrition of that muscle that the hospital cases did worse than the private patients; in the latter more attention could be paid to diet and rest, not only for a week or two, but even for months and years. Those two factors he regarded as of more importance in the care of heart disease in children than the administration of any drug.

Dr. Edmund Cautley said that, in his opinion, the prognosis in heart cases in children varied a good deal according to whether the inflammation was recent or of old standing. Recent inflammations were generally rheumatic, and in them the prognosis did not depend so much upon the murmurs as upon whether or not myocarditis was present. Where there was myocarditis the prognosis as regards recovery was much worse than if there was merely a systolic mitral murmur. And pericarditis affected the prognosis considerably. The prognosis was unfavourable in pericarditis, for it was always combined with a certain amount of myocarditis, and often ended in adherent pericardium. The outlook was most favourable in endocarditis. For many years the tendency had been to base the prognosis on the heart murmurs, but as a rule the heart murmurs could be left out of account apart from any effect on the heart muscle. In cases of old standing the regurgitant murmurs were much less serious than those due to narrowing of the orifices. The outlook in simple mitral regurgitation in children was very favourable; the bulk of them got excellent compensation and lived long. But the prognosis of mitral stenosis was distinctly unfavourable. The prognosis of aortic regurgitation, compared with that of aortic stenosis, was distinctly good; many cases lived to a good age and did hard physical and mental work. In all those cases one had to bear in mind the liability to recurrent attacks. Where there was a strong family history of rheumatism, and the child had had several attacks of that disease, the prognosis was less favourable than if there was a mild attack of rheumatic fever and only mitral regurgitation. The prognosis was worse in recurrent rheumatism, for the poison attacked an already weakened heart. That was especially the case in children, because sometimes there would be very little pain. He frequently saw children brought to the hospital suffering from advanced dilatation of the heart due to myocarditis, generally with mitral regurgitation, where there had not been sufficient pain to attract attention. For that reason rheumatism rendered the prognosis of heart disease in children worse than in adults, for the lack of joint pains led to the rheumatic affection of the heart being overlooked until, perhaps, even fatal damage had resulted.

145. THE DIAGNOSIS OF URINARY TUBERCULOSIS IN CHILDREN.

By CHARLES A. LEEDHAM-GREEN, Ch.M.

(Introduced.)

Our views of urinary tuberculosis have completely changed during the last few years. Instead of regarding it as a relatively rare disease, which when present is difficult to alleviate and hopelessly incurable, we now know that the disease is exceedingly common, and when recognised early and the primary focus removed, of distinctly good prognosis. We know further that the view held for so long and so stoutly defended by Guyon, that the primary disease begins in the bladder and thence spreads along the ureters, causing a secondary infection of the kidneys, is entirely wrong.

Recent investigations on the part of both the clinician and the pathologist have now clearly shown that tuberculosis affecting the urinary tract almost invariably begins in *one* of the kidneys, from whence it spreads to the bladder. Indeed, so far has the pathological view of this disease changed that it is now believed by many (Baumgarten, Hansen, etc.) that there is no such thing as an ascending tuberculosis of the urinary tract, as it is impossible for the non-mobile tubercle bacillus to make its way against the urinary stream. Whether this extreme view is correct or not may be open to doubt, but on the truth and importance of the fact, that the kidney is infected first and the bladder secondarily in the great majority of cases, all are now agreed. The clinical significance of this pathological fact can hardly be over-estimated, for on it to a great extent rests the modern successful treatment of urinary tuberculosis, viz. the early detection and removal of the primary focus.

Importance of early recognition.—The task of diagnosis in a suspected case of urinary tuberculosis is two-fold: (1) The definite proof that the disease is tubercular; (2) the detection of the primary focus of the trouble and the extent to which the disease has spread.

Rarely is any special skill required in determining whether a patient is suffering from tubercular disease of the urinary organs. The localisation of the process may, indeed, call for much experience.

Unfortunately at present men are scarcely alive either to the frequency with which the urinary organs are affected with tuberculosis, or to the significance of the vague early symptoms suggestive of its presence.

Kapsammer* recently examined the post-mortem records for ten years of the Vienna General Hospital, and found that there had been 191 cases where tuberculosis of the kidney had been found at the autopsy. Of all these only

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* 'Nierendiagnostik und Nierenchirurgie Wien.,' 1907.

2 had been rightly diagnosed during life, 4 wrongly so, and 185 not diagnosed at all. Of the 185 undiagnosed in 67 cases only one kidney was involved.

I am often startled by the frequency with which I detect tuberculosis in a patient who, a few years ago before my attention had been drawn to the subject, I should contentedly have diagnosed as suffering from some condition, such as albuminuria of adolescence, essential renal hæmorrhage, chronic nephritis, nocturnal enuresis, etc.

Let me take a case in point :

A little girl was brought to me after having been under medical treatment for some time for chronic nephritis. Her history was as follows :

Some months ago she began to feel languid and to be easily fatigued, and she complained occasionally of a dull aching in her loins.

The urine was acid, perfectly clear and translucent, with no deposit on being left to stand.

On boiling it showed a haze of albumin, and microscopically revealed two or three renal casts and a few blood-cells. It was no wonder that chronic nephritis had been diagnosed.

But on centrifuging the urine and examining the deposit I found numerous tubercle bacilli, and further examination revealed a tubercular focus in the left kidney.

Again, a healthy-looking boy, aged 12 years, was brought by his mother because of the frequency with which he wet the bed at night. He had been treated elsewhere without avail ; indeed, he was worse rather than better, in spite of having been circumcised, sounded and dosed with belladonna and worm-powders, the mother finally being told that the trouble was of nervous origin and would disappear at puberty.

On examining the urine I found it highly suspicious, for it was faintly opalescent, and acid ; and the centrifuged deposit showed a fair number of pus cells and an absence of micro-organisms when stained with methylene blue.

On being stained appropriately tubercle bacilli were found without difficulty.

I have quoted these two cases, not because they are of special interest or in any way remarkable, but because they illustrate the importance of two points in the diagnosis of tubercular disease of the urinary organs, viz. firstly the attention which should be given to the slightest disturbance, be it discomfort or undue frequency, in the act of micturition, and secondly, that the routine examination of a urine should include a *bacteriological* as well as the ordinary chemical and microscopical one. For it is in this way, and this way alone, that the suspicion of tubercle is likely to be raised whilst the disease is still in its early stages and most amenable to treatment.

The systematic examination of the urine of all patients suffering from albuminuria, irrespective of whether pus or blood is present, will reveal the presence of tubercle in a surprising number of unsuspected cases.

It is often stated that tubercle bacilli are difficult to find in the urine, and that they are not detected in more that 25 per cent. of such cases. This is probably true, but it is only so because the urine is imperfectly examined. If the total quantity of urine passed in twenty-four hours be mixed with a little carbolic acid and be allowed to stand for a day in a tall cylindrical vessel, and then the deposit be centrifuged and a number of stained films examined, it will be, I believe, but rarely that the specific bacilli cannot be detected. Rovsing found them in over 80 per cent. of his cases, and I do not think that figure is too high. A suspicion of tuberculosis should always be awakened when, in spite of pus cells being present in the urine, none of the usual pyogenic microbes are found. If, in spite of long and repeated examinations, no bacilli can be detected in the urine of a suspected case, then a small quantity of the urinary sediment may be injected subcutaneously or intra-peritoneally into a guinea-pig, and the animal killed a few weeks later with a view to seeing whether tubercular infection supervened or not. Under proper control-experiments this test is reliable, and may prove of great value.

Bloch advises the subcutaneous crushing of the inguinal glands of the guinea pig prior to the injection of the suspicious urine in their neighbourhood. Should the urine contain tubercle bacilli in two or three weeks' time the crushed glands will show the characteristic lesions and the specific micro-organism.

In cases of doubt resort should be taken both to the estimation of the opsonic index of the blood and to Calmette's conjunctival or v. Pirquet's cutaneous reaction to tuberculin.

I have made use of these diagnostic tests freely, and have often received great help from them. Calmette's conjunctival reaction especially I have found most valuable and far more reliable than the opsonic index. It is true that the absence of the reaction does not necessarily exclude the presence of tubercle, especially of advanced general tuberculosis, and, on the other hand, the reaction is occasionally (never in my own cases) met with in typhoid fever and other non-tubercular conditions. Yet this does not render the reaction valueless as has been stated, any more than the occasional failure of the Widal reaction stamps it as useless. It is only those who seek for an infallible sign who will be disappointed with the reaction. Two precautions are advisable, firstly, to begin with a weak solution in each case, advancing to the stronger solution if no reaction takes place; secondly, never to employ the test if there is evidence or history of inflammation of the eye. When for any reason it seems undesirable to make use of the conjunctival reaction, the cutaneous test of v. Pirquet may be employed. It seems to be equally sensitive and is devoid of the slightest risk.

The weak point about the opsonic index and the ophthalmic and cutaneous reactions is that—(*a*) they give little, if any, idea of the severity and extent of the tubercular infection—an insignificant quiescent nodule in a lymph-gland, it may be, often giving rise to a marked positive reaction; and (*b*) they in no wise indicate the locality of the illness.

The only reaction which gives us a clue to the site of the infection is that produced by the subcutaneous injection of Koch's old tuberculin. This test has, I think, undeservedly fallen into disrepute as being dangerous, from its liability to fan into flame a latent tubercular focus. There is no doubt that much harm was done some years ago by its indiscriminate and excessive use, but there is evidence to show that if used cautiously in suitable surgical cases it is a highly valuable diagnostic agent, for the reaction which follows its use frequently, though by no means always, indicates through localised pain or tenderness the site of the trouble. In tuberculosis of the urinary organs the injection often produces such a temporary alteration in the character of the urine by the presence of pus cells and tubercle bacilli as to clear up all doubt as to the nature of the illness.

Having found tubercle bacilli in the urine, or otherwise being satisfied that the suspicion of tuberculosis of the urinary organs is justified, the next step in the diagnosis is the detection of the primary focus and the determination of the extent of the disease.

This is an essential step in the modern surgical treatment of urinary tuberculosis, and upon the accuracy with which it can be determined largely depends the success of the treatment. Happily, owing to recent improved methods of urinary diagnosis, a great refinement in diagnostic power has been attained, so that we are not only able in the majority of cases to detect the primary focus, but even to estimate, with surprising accuracy, the extent of the disease. Having found tubercle bacilli in the urine the next point is to determine the condition of the two kidneys and the bladder, for, for the purposes of this paper, we may ignore the possibility of the primary focus starting in the testicle (the detection of which offers no difficulties) and in the ovary or tube, which from anatomical reasons is exceptionally rare.

In practice the problem is narrowed down to this: (1) Is the bladder involved; if so, how far? (2) What is the condition of each kidney?

Before these questions can be fully answered a thorough and methodical physical examination of the urinary organs is essential. It is unnecessary to enter here into all the details of such an examination, and I must confine my remarks more especially to those points where the technique in dealing with the child differs from that as applied to the adult.

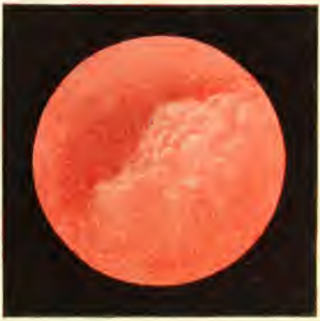
Special attention should always be paid to the condition of the ureter. If the kidney is tubercular the patient will generally complain of pain on pressure being made over the course of the ureter, more especially at the hilum of the kidney, at the point where the ureter passes over the brim of the true pelvis and at its entrance into the bladder. The lowest portion of the ureter is directly accessible to palpation by the finger inserted *per rectum* in the male and *per vaginam* in the female. In the normal condition it is rarely possible to distinguish the form of the ureter, but when affected with tuberculosis it can almost always be felt as a cord-like structure, indurated and sensitive.

The extent of the involvement of the bladder may be roughly gauged by its tolerance of distension. If the urine can be retained for some hours without discomfort, or if the bladder can be moderately distended without giving rise to tenesmus or hæmorrhage, it is improbable that there is any extensive tuberculosis of that organ. But to gain anything like an exact idea of the condition of the urinary organs a cystoscopic examination is essential.

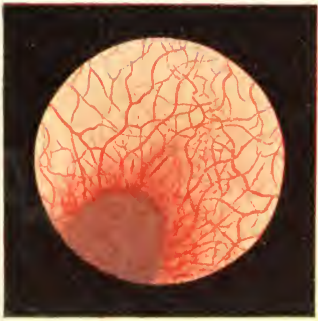
Thanks to the excellent optical arrangement of the modern cystoscope, by which even with so fine a calibre as No. 14 or 15 (French scale) a clear field of vision is obtained, simple cystoscopy can be practised in the youngest girls and in boys over eight years of age.

In the majority of cases the bladder will show some signs of the disease. This holds true not only in those

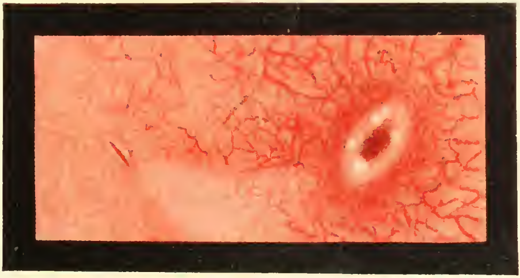
PLATE XVII.



A characteristic early tubercular infiltration, as seen through the cystoscope.



A large tubercular ulcer below the orifice of the right ureter.



Cystoscopic view of the base of the bladder in a case of tuberculosis of the left kidney (Wyatt). The opening of the right ureter is normal; the opening of the left ureter is seen to be gaping, the lips œdematous and thickened, showing the presence of small miliary tubercles.

Mr. Charles A. Leedham-Green's Paper on The Diagnosis of Urinary Tuberculosis in Children.

Adlard & Son, Impr.

cases of relatively advanced tuberculosis of the kidney where the infection has spread down the ureter and involved the bladder, but also in those cases where the mischief is still confined to one kidney and there is an absence of all bladder symptoms. In the former, tubercular nodules and ulceration will be seen grouped for the most part about the opening of one of the ureters.

In the early stages of kidney tuberculosis the bladder may be quite free from ulceration or gross lesions of any kind, but it is rarely that a careful inspection of the opening of one of the ureters does not show some indication of the trouble, such as hyperæmia and inflammatory œdema of the lips of the opening, pouting, gaping, or retraction of the orifice.

If the tubercular lesions are grouped wholly or largely round the mouth of one ureter, the rest of the bladder-wall being healthy, there is strong presumptive evidence that the kidney on that side is the origin of the trouble. I say "presumptive" evidence, because although as a rule subsequent investigation will prove this to be true, yet every now and again it is found that the lesion in *the bladder* in most marked on the *opposite side* to the affected *kidney*. Or, although both kidneys are severely infected, the bladder trouble is localised round one ureter only. And again, and not infrequently, though only one kidney is affected, the whole of the bladder is ulcerated. I am therefore amazed when I read of surgeons trusting to a simple cystoscopic examination to decide as to the condition of the two kidneys.

A valuable adjunct to the simple cystoscopic examination is the so-called chromo-cystoscopy, which consists essentially in the observing by means of a cystoscope the excretion by the kidneys of a pigment (carmine blue) injected subcutaneously. If the bladder of a healthy person is cystoscoped a few minutes after the subcutaneous injection of the pigment and the opening of the ureters be watched, a jet of blue-black urine will be seen to be squirted from time to time into the bladder by the peristaltic contractions of the ureter. If there be any mechanical or functional interference with the secretion or excretion of the urine, then the excretion of the blue pigment will be delayed, diminished in amount, or absent altogether. If the urine is seen to be entering the bladder in regular forcible squirts of dark blue fluid from both ureters the presumption is that both kidneys are healthy. If, on the other hand, the blue jets are seen on the one side only, and from the other ureter issues only faintly stained, or clear fluid, the probability is that the disease of the kidney or ureter is on the side of the unpigmented urine. If the excretion of blue is very faint, greatly delayed, or absent altogether on both sides, then the suspicion is raised that both kidneys are diseased.

For lack of time I have been obliged to state this very crudely. There are many points to which attention must be paid before drawing important conclusions from the chromo-cystoscopic appearance. But, with all its reservations and possible fallacies, chromo-cystoscopy is of great service in the diagnosis of tubercular disease of the kidney. It rarely fails to indicate which kidney is the chief seat of the disease and from which the infection of the bladder has sprung. And it gives some indication, though not a very fine one, as to the amount of destruction which has taken place, and the power of functional activity which remains. Too much reliance must not, however, be placed upon it, for though gross lesions are generally clearly indicated, small though serious ones may not be shown. Thus, because the blue pigment is seen issuing from both ureters, it does not necessarily mean that both kidneys are sound, or free from tubercle. As an adjunct to our diagnostic measures it is most valuable, but it lacks the necessary delicacy, which it is so desirable to obtain when deciding whether nephrectomy is permissible or not. The only way in which a really accurate knowledge of the condition of the kidneys can be gained is by collecting the urine as it is secreted from each kidney separately.

In the adult there is, I believe, only one method by which this can be done worth consideration, and that is the catheterisation of both ureters by means of the cystoscope. And this is certainly the method of choice in the case of the child where the size of the urethra will permit the introduction of the instrument. Unfortunately, the finest catheter-cystoscope which can be made (no. 18 French) is too large to permit of its passing down the urethra of a boy under twelve or thirteen years of age. Consequently there are many cases where the catheterisation of the ureters cannot be undertaken in children, and reliance must be placed upon other measures. In the majority of such cases I find that the results of chromo-cystoscopy, taken together with such data as may be gained by noting the daily excretion of urea, the freezing point of the blood, and the interval which elapses between the subcutaneous injection of phloridzin and its first appearance as sugar in the urine, not only indicate the site of the tubercular lesion, but also the functional activity of the kidney with sufficient accuracy as to enable one to decide whether a nephrectomy is feasible or not.

In some cases, however, one sees numerous tubercle bacilli in the urine with perhaps an occasional attack of transitory hæmaturia, where neither the simple nor the chromo-cystoscope nor palpation enables one to say on which side the lesion is present.

To decide the point an exploratory nephrotomy may be made, but it is an operation of considerable severity for a child, and may be attended by severe hæmorrhage difficult to arrest. A much simpler procedure, and one which I have found entirely satisfactory, is to expose the ureter subperitoneally by a small incision in the semi-lunar line of the abdominal wall, open its lumen by a small longitudinal cut and insert a fine urethral catheter, and so collect the urine separately. When the examination is completed a fine stitch closes the opening in the ureter, which is then dropped back into its place and the wound drained for a day or so.

Provided the urine from the two kidneys has been collected separately, there is rarely any difficulty in determining both the site and the extent of the disease and the likelihood of operative interference proving successful.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th*, 1908.)

146. CERTAIN TYPES OF FEEBLE-MINDED CHILDREN AND THEIR SIGNIFICANCE.

By W. A. POTTS, M.D.

Some aspects of the problems relating to feeble-minded children would seem to be a suitable subject for a paper at the meeting in Birmingham of this Society. In connection with defectives Birmingham has always taken a forward part. It was one of the first towns to institute special schools, and its After-Care Committee, which concerns itself with the care of those children after leaving school, was the first of its kind. The Sandwell Hall Boarding School for mentally defective children was the second of such schools in point of time, while the Monyhall Colony for Epileptic and Feeble-Minded Persons is the first institution established by guardians for uncertified cases, and represents the first combination of boards of guardians to deal with these cases in a manner none of them could have undertaken singly. Birmingham has supplied a large amount of evidence for the Royal Commission now sitting on this subject, and was selected by the Commission as one of the areas for a special inquiry. While work of such great practical importance has been accomplished scientific investigations have not been neglected. Observations made in the special schools and other institutions here form to a large extent the basis of this paper.

For some years it has appeared to me that there is a close connection between the type of a feeble-minded child and its origin. There is nothing extraordinary in this idea. The merest tyro often recognises the offspring of tubercular and syphilitic stock. Why should not our discrimination in such directions be extended? All observers recognise a certain number of different types of these children, which are readily distinguished; why should not some of these types have a definite heredity?

The type, too, greatly influences the prognosis; every expert looks for certain characteristics, which he considers favourable or the reverse. In regard to these children the prognosis is of peculiar importance. Many cases are brought to me, not so much for a diagnosis, for that is often obvious, but for an opinion as to their prospects. Will they ever improve, and if so to what extent? Is it worth while spending any money at all on education? Can they ever be self-supporting? If emigrated is there a reasonable chance of their holding their own and keeping out of the hands of the police? No details that will help us to answer such questions are too insignificant.

First let us consider the Mongolian type, so called from its resemblance to the races in the far East. This group is readily distinguished by certain characteristics in the shape of the head, the eyes, the tongue and the hand. The forehead is rather square, and the skull is of a short, oval shape, its breadth approximating to its length; the ear tends to a similar pattern. The eyes are almond-shaped and obliquely set, with an epicanthic fold. There is a squat nose. The tongue shows transverse furrows. The hands are broad, and the fingers short, especially the little finger, which is frequently incurved. The skin is thick and coarse, often eczematous, with a tendency to a bronze discoloration, and there may be a growth of hair in an unusual situation. Such are the marks of a typical Mongolian. Well-marked cases are, however, rare, as I soon remembered when looking for one to show this afternoon; less characteristic individuals with some of the features, such as the furrows in the tongue completely absent and others somewhat masked, are not uncommon. Can we associate anything definite with this type? Some years ago Dr. Shuttleworth expressed the opinion that such are essentially unfinished children, and that their peculiar appearance is really that of a phase of fœtal life, some defect of formative force being usually traced in connection with their intra-uterine life, not uncommonly ill-health of the mother. He further stated that nearly half these children are the last born of a long family, when the procreative powers are at a low ebb, while in many there is a family history of phthisis. My own observations confirm these statements except that I have found the type to be at least as frequent in the first-born of a family as in the latest born; in a large group of defective cases containing eight Mongolians five were the eldest of the family. They are seldom found among the next three or four, but are certainly often at the tail; one of my cases was the seventeenth. This coincides with the observation that the eldest is frequently not the most robust of the family, that the second and third are the healthiest, the fourth and fifth being also usually up to as good a standard, while after the fifth the progeny gradually deteriorates. A family history of phthisis is without doubt usually to be found in association with this type, but I do not regard phthisis as the essential ætiological factor; the prime cause appears to me to be poor physique of the mother. People of poor physique are frequently the victims of tuberculosis; hence the usual, but not necessary, association with tuberculosis. This type is not uncommon in the better classes, occurring as the offspring of women who are really not physically fit to have children at all. Two instances at once occur to me: one of these, a women who never menstruated till she was five-and-twenty, consulted a physician as to whether this was any bar to matrimony; he saw no objection, but the subsequent birth of a typical Mongolian suggested that the amenorrhœa might have been looked upon as an indication of a want of procreative force. In the other instance a couple of highly neurotic parents had five more or less normal children; the sixth, born when the mother was well over forty, was an ament of this group. The decision that a child conforms to this type also influences the prognosis; though many are bright in some ways and full of promise, they seldom accomplish much; their physique is nearly always poor; the majority die of tubercular disease before reaching adult life.

I wish next to discuss the type spoken of as the simple congenital. These show no marked deformity of head or limbs. They are usually below the average height with heads of normal shape, but the expression of the face is vacant, and there are several obvious anatomical peculiarities—the so-called stigmata of degeneration. In making a diagnosis an important point to remember is that the stigmata tend to be multiple in defectives, instead of occurring singly, as may happen in normal individuals.

The abnormalities are chiefly found in the face, head, and hand, the group, well described by Fletcher Beach, being without much difficulty quite definitely marked off.

The irregularities take the form of obliteration or exaggeration of normal markings, such as the antihelix or other parts of the ear, or consist in marked diminution in size of such important landmarks as the mouth, orbital fissures, or lower jaw. The teeth are often irregular, and may be arranged in two rows, while the ear is implanted too far back.

In my experience this type is pathognomic of an insane or feeble-minded ancestry; mental incompetence is their birth-right. In the group of cases already referred to there were 24 who fell into this class; 15 of these were ascertained to have such a heredity. This gives a percentage of over 60, which is higher than that obtained among the feeble-minded as a whole. In a group of 250 consecutive cases of feeble-minded children in which I made special inquiries into the family history, only 30 per cent. had a similar history. In some of these cases there is a continuous line of defective ancestry; thus both the mother and the grandmother of one boy I showed this afternoon are obviously feeble-minded. When the public at last recognises the necessity of segregation this class ought to be considerably diminished. As regards prognosis in children of this type I find their physique often to be fairly good, and their expectation of life longer than obtains among the feeble-minded as a rule. I can also confirm Fletcher Beach's statement that "these cases, unless of a very low type, improve very much under proper education and training."

Next take the neurotic type, quite a distinct one, though the classification is on a different basis, for it may include members of the Mongolian, hydrocephalic, or other groups distinguished by the cranial configuration. The chief characteristic is weakness, mental and physical. The nervous system is both weak and irritable in marked contradistinction to the stolidity of some defectives. The power of attention is almost *nil.* The eyes quickly wander if directed to a fixed object. If asked to extend the arms in front at the shoulder level the arms are not raised to that level, nor are they straight, and Warner's "weak hand balance" is noticed. "The wrist droops, the bones of the palm of the hand are somewhat folded together, while the thumb drops and all the fingers are slightly bent." Often the left hand is kept at a lower level than the right. What is the significance of this type? I have little doubt that it is pathognomonic of an alcoholic heredity. A history of alcoholism was obtained in 40 per cent. of the group of 250 defective children in which I inquired specially into the family history. The majority of the 40 per cent. were included in this type. I have been told by other workers in this direction that they have noticed this connection between a history of alcoholism and the nervous group. This is not inconsistent with the selective action of alcohol as a poison on nerve-tissues, and is the human corroboration of the experiments made in Germany with dogs. If a pregnant bitch is treated with alcohol the litter tend to develop epilepsy and other signs of mental weakness.

I believe careful observation and full inquiry would result in definite knowledge with regard to other types. For instance, we can probably confirm what Shuttleworth has said of the microcephalic, that the prognosis is favourable or otherwise in proportion to the size of the head. It is well to remember that this only holds good till the size of the head approaches to the normal, for in the macrocephalic or hypertrophic cases, as they are sometimes called, the larger the head the more stupid the child. The unfavourable element therefore is the extent of deviation from the normal. In this connection it is interesting to note the impression with regard to the group of defective children who are normal in appearance, well developed, and often good looking, and who can only be distinguished by an intellectual examination. The prognosis here is bad; in the words of Langdon-Down, it is "inversely as the child is comely, fair to look upon and winsome."

The last type which I wish to describe is the moral defective. This again is based on a different classification, our guide being the social conduct. It would never do to explain many cases of wrong-doing by inclusion in this group; as a matter of fact, my observation in prisons has convinced me that only very few of those found there can plead this abnormality as a justification. Still, I believe the type does exist, and that its recognition is important. Just as an instance, consider a youth, aged 19 years, who was brought to see me recently; he is the son of ordinary respectable people. He was educated at an ordinary school, and subsequently at a technical school, where he exhibited special talent, especially for drawing. He writes an exquisite hand, and is an expert in photography. He has had several situations, usually as a clerk, but if he has not been summarily dismissed, he has always given them up after a few weeks' work. He was a thief from an early age, and has frequently been caught in the act at school and elsewhere. He has several times been in the hands of the police, and has served a term of imprisonment. He is utterly depraved; he lies in bed in the morning, and spends all the money he can get on drink and vice; he steals from his own family, and pawns the clothes with which he is provided. Although he will not do ordinary work he spends hours in his favourite hobby of enlarging photographs; this he does, not in the usual way, but in a manner suggestive of a mental twist, for he makes a large pencil drawing of a small photograph, and then photographs the large drawing. So accurately is this done that anyone would believe the enlargement to be effected in the usual way. He brought several specimens for me to see, and I could only think that his prospects as a forger were far too brilliant. He is certainly abnormal, for he has a narrow forehead, suffers from an extreme degree of myopia, and has a slightly dilated heart. When I carefully studied ninety-seven consecutive cases admitted to a Magdalen home, I found that seven of these might be fairly described as morally defective. This home, I may explain, is filled with young girls, practically all of them in their teens. I classified as morally defective girls who were sharp and intelligent, but without sense of honour or modesty, and who were insusceptible to moral and religious training, thereby differing markedly from the majority; nothing could restrain them from lying and from stealing from their companions. I could quote many other instances, but must not say more now than that essential features in the diagnosis of these cases are the consideration that the crime is out of all proportion to the temptation, and further, that the moral shortcomings are not to be explained by training and environment. These cases are very difficult to deal with, though something may be accomplished by hygiene and prolonged training.

The subject of this paper is one to which it is difficult to do justice in a limited time. If I have brought forward any evidence in favour of the value of carefully studying different types and noting their characteristics, my object in writing it has been achieved.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th,* 1908.)

147. THE TREATMENT OF CONGENITAL DISLOCATION OF THE HIP.

By FRANK BARNES, M.S.

(Introduced.)

The treatment of congenital disease of the hip as the subject of surgical discussion has been dealt with so frequently that I feel I owe you an apology for its introduction, but my excuse is that apparently some scepticism still prevails among both surgeons and physicians as to the value of the methods introduced to us by Hoffa and Lorenz, and which they have done so much to elaborate.

In the short time at my disposal I can only emphasise the factors which I consider of the most importance in the treatment of this condition, and shall doubtless lay myself open to the charge of having neglected many details both of pathology and treatment which make for success or failure, but which I shall hardly be able to mention.

It will suffice me if I can prove to your satisfaction that it is upon the early recognition, and before all upon the early treatment of this condition, that we obtain the best results from a routine, one of the most tedious in surgery, and one from which we can hardly look for practical results before some twelve or eighteen months have elapsed from the onset.

May I briefly mention some of the more interesting details regarding the condition and its history:

Discovered and described by Dupuytren in 1826 it was for a long time considered to be incurable, and probably for one or two reasons. In the first place it was rarely discovered until the secondary deformities had become marked, and then the treatment practically consisted in applying extension in order to draw the femur down to its natural position.

Then, again, it was considered that the primary lesion lay in a defect of the acetabulum, or rather, I should say, in the want of development of the acetabulum. And this being so, no treatment could be of any avail.

In 1890 the first open operation was performed by Hoffa, of Berlin, who opened the joint by a posterior incision, deepened the socket by means of gouges and then replaced the head of the femur with good results in a limited number of cases, but many things were against it. Owing to the depth of the structures it meant a serious loss of blood, many muscles had to be cut, and the mortality from the operation itself was very high. Moreover, its results were usually unsatisfactory, relapses being the rule, and where the hip remained in position ankylosis frequently occurred, but certain observations were made which have led to the modern treatment. It was found that an acetabulum of fair depth and development was common especially in the younger cases. This fact has been verified of recent years through the agency of the X rays, which have undoubtedly contributed in the highest degree to the adequate understanding of the methods of cure. The open operation also demonstrated another fact, since confirmed by the radiograph, and that is, that in old-standing cases the head of the femur frequently developed a depression on the ilium, a sort of secondary acetabulum in fact, which to some extent acts as a socket for the head, and also limits its movement upwards.

On examination of radiographs of various cases it will be seen that the acetabulum varies very considerably, in some being apparently of normal size and in others very shallow and bearing more resemblance to the glenoid cavity of the scapula, this difference being more marked as compared with the normal in the older patients. In fact, the younger the patient the less likelihood of there being any gross difference in the cavities of the two sides.

This has been carefully worked out by Heusner and Marewald, who on the examination of a large number of fœtuses made these observations, that the acetabulum is relatively shallow in fœtal life, that in new-born children covers one third of the head of the femur, whereas in children five years of age one half of the head is covered by the acetabulum. Also, that there was a disproportionate laxity of the capsule in female as regards male, and that therefore in all probability the primary condition was a subluxation only, which became complete on standing or walking. This laxity of the capsule probably accounts for the prevalence in females, 84 per cent. being of that sex.

It seems, therefore, that the depth of the acetabulum varies with age, or, to put it in another way, that it depends for its normal development upon the apposition of the head of the femur, and this is the fact around which centres the modern treatment of congenital dislocation. The marked projection of the upper lip of the acetabulum with its buttress of bone running into the anterior inferior spine is a development consequent upon the transmission of the weight of the body through the ilium on to the heads of the femur, and, therefore, an essential part of the treatment is that after reduction the child should be made to walk with the femoral head in its proper position.

When several years have elapsed before treatment this buttress is wanting and attempts at reposition naturally fail.

I have said that in very young patients there is relatively in the majority of cases very little difference in the acetabular cavities of the two sides, and probably until the child commences to walk there is nothing to call attention to the condition; the head of the femur, lying in close proximity to the joint, being pushed upwards and backwards by the act of walking. At this stage, therefore, reduction is easy. Later on other changes take place which militate against reduction, such as the atrophy of the head of the femur, obliquity and shortening of the neck, and what is more important from the immediate point of view of reduction, changes in the capsule. This becomes stretched out like a strap over the cavity and often develops a sort of hour-glass contraction, offering an almost effective barrier to the reduction by manipulation.

It is impossible to over-estimate this advantage of age in the light of treatment. Not every case of congenital dislocation can be cured, but if diagnosed early probably the majority can be successfully dealt with, whereas after a certain age very few indeed are amenable to treatment, and this age I am inclined to make five or six years. This, I know, is somewhat lower than many other surgeons go, but I refer to actual cure, where the head of the femur lies in its proper cavity and is not merely an anterior transposition so frequently vaunted as a cure. The main factors against success are the changes that take place in the whole of the structures concerned consequent upon this false position, and it is imperatively necessary that treatment should be adopted before these secondary changes become established.

I cannot in the short space of time allotted me go into minute detail of the various steps in the reduction of the dislocation, and I shall merely mention shortly a few of the difficulties I have met with. In the first place by manipulation in various directions the resistance of surrounding structures has to be overcome. These are, as has been already pointed out, the more dense and resistant the older the patient, while in cases which have not long walked, *i. e.* in children up to the age of two or three, reduction may be comparatively easy.

Where a well-marked acetabulum exists with a distinct margin or buttress superiorly, the difficulty of levering the head over the posterior lip may be great, but these cases in my experience have a very good prognosis, especially where the head remains in the socket in the flexed and abducted position without assistance. Those cases in which the head slips easily into the joint, but as readily slips out owing to a deficient posterior lip and perhaps a tight anterior portion of capsule, have not nearly as bright an outlook, since it is extremely difficult to keep the limb in position ; no matter how well-fitting the case, it always allows of some shifting of position on the part of the child, and, therefore, a loss of the proper relationship between the head and the socket.

I have found that when the obstacle to reduction lies in a contracted anterior capsule which levers the head out as soon as in position, a good plan is to fix the limb temporarily for a week in the flexed and over-abducted position. This stretches the tight portion and reduction becomes much easier on the second attempt.

Where the posterior lip is wanting the prognosis is bad, since these cases are usually older and the acetabulum is too shallow to adequately support the head, and probably the better treatment would be to convert the posterior displacement into an anterior one. This results in a much better functional state, the child walks more uprightly, the lordosis dissapears to a marked extent, and there is less shortening.

Some surgeons as a preliminary to reduction employ heavy weight and extension, even up to 30 or 40 lbs., for a week or fortnight beforehand. This is probably of more value where the capsule has become markedly contracted, though probably of little use in the variety known as hour-glass contraction.

Frequently, however, it becomes necessary to use the tenotome to overcome resistant structures, and especially with regard to the adductor muscles, which have become shortened. These not only oppose reduction, but act as a lever pushing the femur backwards, hence it is sometimes advisable to cut the adductor longus near its insertion into the pubes in order to ensure greater stability during the first month or so of treatment.

After reduction, with its great bruising of the parts, and the subcutaneous tearing of various structures which is most frequently necessary, the limb is placed in what is known as the frog position, flexed at right angles or even beyond this, and abducted to the full extent. It is fixed in this position by means of a plaster case and as soon as possible the child encouraged to walk. In my own practice I, however, never allow walking in the original case for this reason: there has been a considerable interference with the structures of the part; portions of the capsule which were stretched are now loose and *vice-versâ*, so that for the first month or five weeks the patient is simply kept in the recumbent position until the various structures have settled down and become accustomed to the new position. Then a new case is applied and the child begins to walk either by means of a high boot or a patten, or, in the case of double dislocations, with a stool or chair. This position is maintained for six or eight months and then the limb is gradually brought down, still being held by means of plaster cases until the child is able to walk almost naturally. During this time it is very necessary to have occasional radiographs taken so as to ascertain the position of the head, and especially during the first few months.

It is this ambulatory treatment which constitutes the essential feature of the modern method. The socket is deepened by the natural process of the pressure of the head of the femur, and this is the lesson we have learned from the secondary hollows developed in old-standing cases and from the natural growth of the normal acetabulum. The probability is that in the majority of cases the acetabulum is not to blame in the first place, but that the original cause is some malposition *in utero* combined with an abnormal laxity of the capsule, and that the acetabular changes are due to a secondary and not primary lack of development.

Some difference of opinion exists with regard to the best way to apply the plaster case. My own method is to cover the parts involved with a thick layer of brown cotton-wool, which is then fixed firmly by a flannelette bandage, the main point being to take precautions lest any sores from pressure or rubbing should be caused. And in the first two or three cases I include the opposite thigh slightly abducted; this I find gives much better fixation; at the same time I strengthen various naturally weak spots, such as the folds of the groin, by extra layers of bandage. And at the back of the affected hip I include a roll of plaster held in position until set so as to prevent the liability to displacement backwards which is very difficult to counteract. The cases are changed once every six weeks, not only on account of cleanliness, but also that I may examine the condition of the joint, take a radiograph occasionally, and vary the position according to circumstances.

In certain cases a cure may be looked forward to in about twelve or eighteen months, and in a few all apparatus may now be discarded, but in a certain number of the cures a hip splint in the form of a pelvic band and thigh support should be used for some two or three years afterwards.

In conclusion, let me assure you that this short paper is not intended to be even a summary of the various conditions governing either the treatment or the pathology of congenital dislocation of the hip, but merely a plea for the better understanding of the paramount importance of two factors—early recognition and early treatment.

(*Read at the Provincial Meeting held at the Children's Hospital, Birmingham, Saturday, June the 20th,* 1908.)

Discussion.

Mr. A. H. Tubby said that as the time for the meeting had almost expired he would be very brief. He complimented Mr. Barnes on an excellent *resumé* of the subject, and on most points he, Mr. Tubby, was in accord with the author. If he were asked what were the three most important points in the prognosis of those cases he would say: first, the age; secondly, the condition of the acetabulum, especially as to the development of the posterior lip or not, and thirdly, the shape and outline of the head and neck of the femur. With regard to the position which the head took in cases of congenital dislocation, it was usually stated that the head was almost always posterior. His own experience latterly had been perhaps nearly unique, as the majority of dislocations he had seen were anterior, and he suspected that in some of those cases a natural transposition had taken place from posterior to anterior. The difficulties in the treatment of the dislocation were really immense, and what was still wanted, he thought, were good tests of actual replacement of the head. The X rays could not be entirely relied on in that respect, as the head might be slightly in front of or slightly behind the acetabulum, and the shadows then overlapped. He had often seen cases shown as cures, but he had his doubts on that point. He considered that a cured case must, to be so regarded, fulfil certain conditions. That which he regarded as most important was the position of the head in relation to the femoral artery. The central point of the head of the femur should lie almost exactly underneath the femoral artery as it passed through Scarpa's triangle. In many cases of anterior transposition the head was nearly in that position, but it would be found to be much too high in the groin. Such cases were not completely cured. The heads of the femora must also occupy the same relative positions in the two thighs. Then, having ascertained that the head of the femur was in good position, it must be stable. The best way to ascertain that was to take hold of the thigh low down, ask someone to take some of the weight of the pelvis by lifting it, and then, with the head pushed firmly into the socket, the pelvis and femur should move as one.

On the proposition of **The Chairman (Dr. George Carpenter)** the meeting passed a cordial vote of thanks to the Chairmen and Committees of the General Hospital and of the Children's Hospital, Steelhouse Lane, for their kindness in allowing the Society the use of their splendid institutions.

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